

INTRODUCTION TO DERMATOLOGY

Intended Learning Outcomes

- 1. Introduction.**
- 2. Normal skin histology.**
- 3. Functions of the skin.**
- 4. Primary & secondary lesions.**

The Skin

- Largest organ of the body.
- 1/7 of body weight.
- Surface area is 1.75 m².
- Examination of the skin is always part of the clinical examination of the body, e.g. pallor & cyanosis.
- Skin diseases may have associated internal organ involvement, e.g. psoriasis & arthritis.
- Cutaneous findings can be a clue to internal diseases.

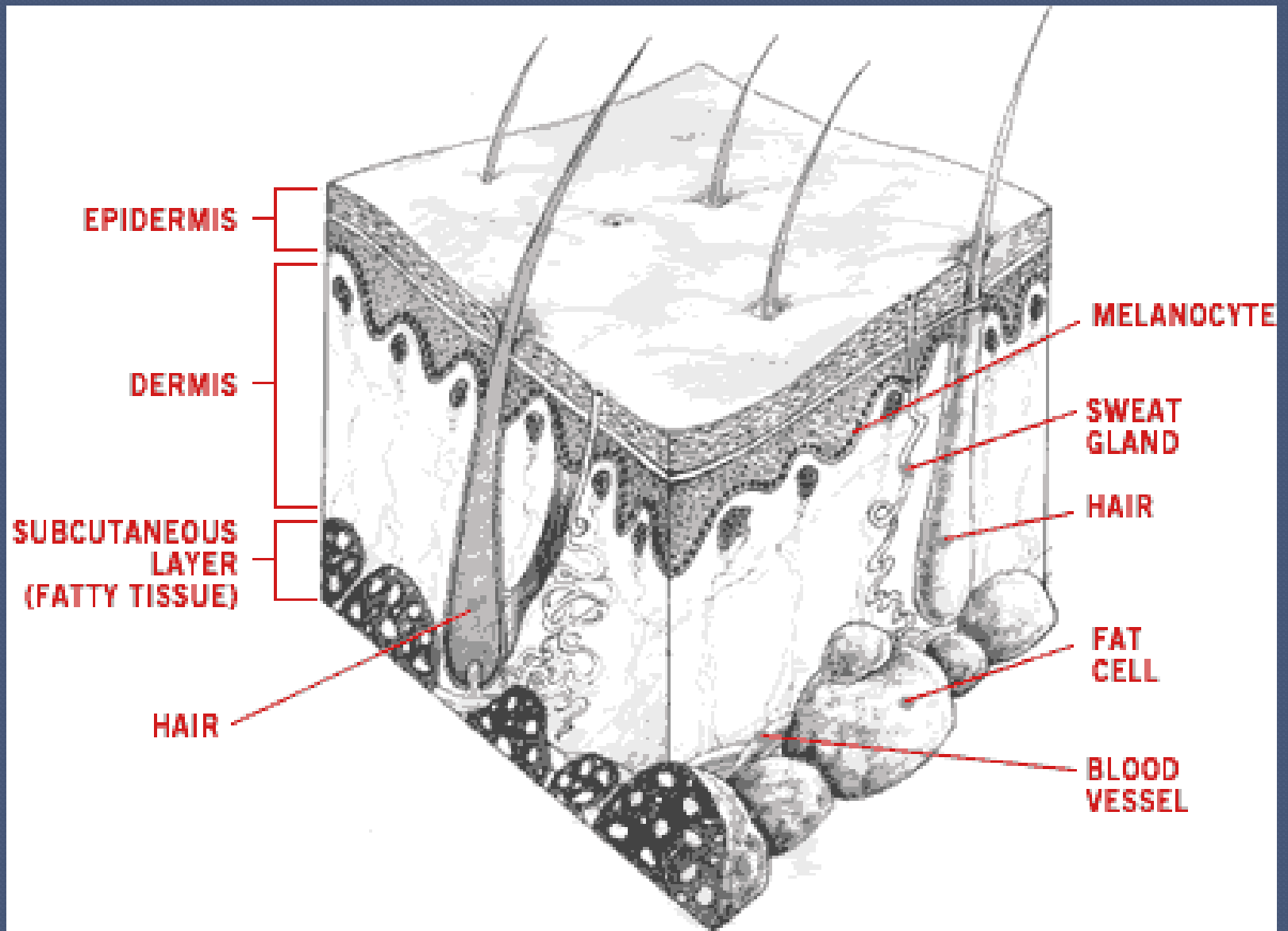
Management of Skin Disease

1. Complaint.
2. History.
3. Examination.
4. Investigations, e.g. skin biopsy.
5. Treatment.

Structure of Skin

Skin is composed of 3 main layers

- I. Epidermis.
- II. Dermis.
- III. Hypodermis.



I. Epidermis

- Outermost layer.
- Rests on the basement membrane.
- Formed of cells.

Keratinocytes (KCs):

For formation of keratin.

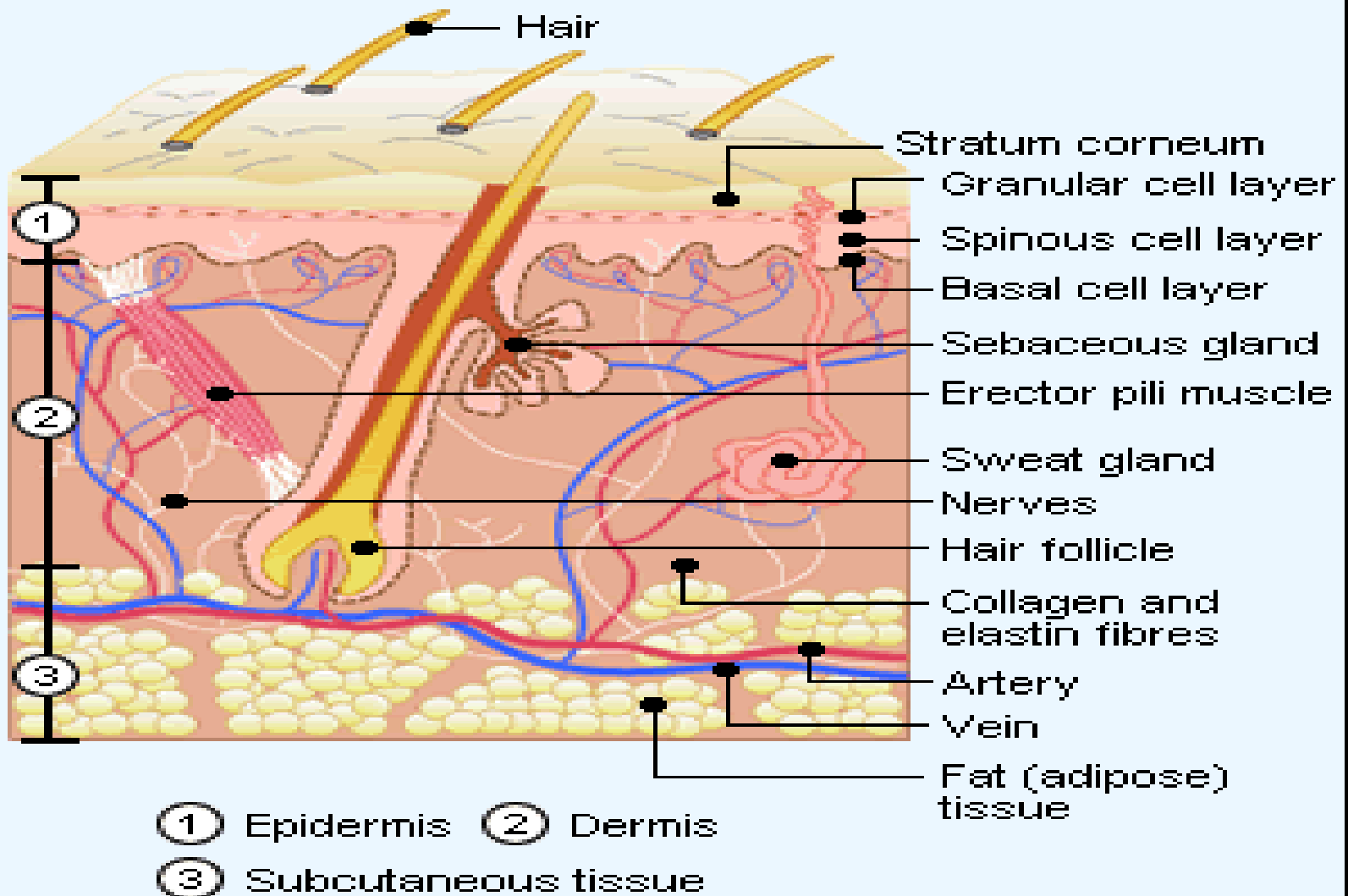
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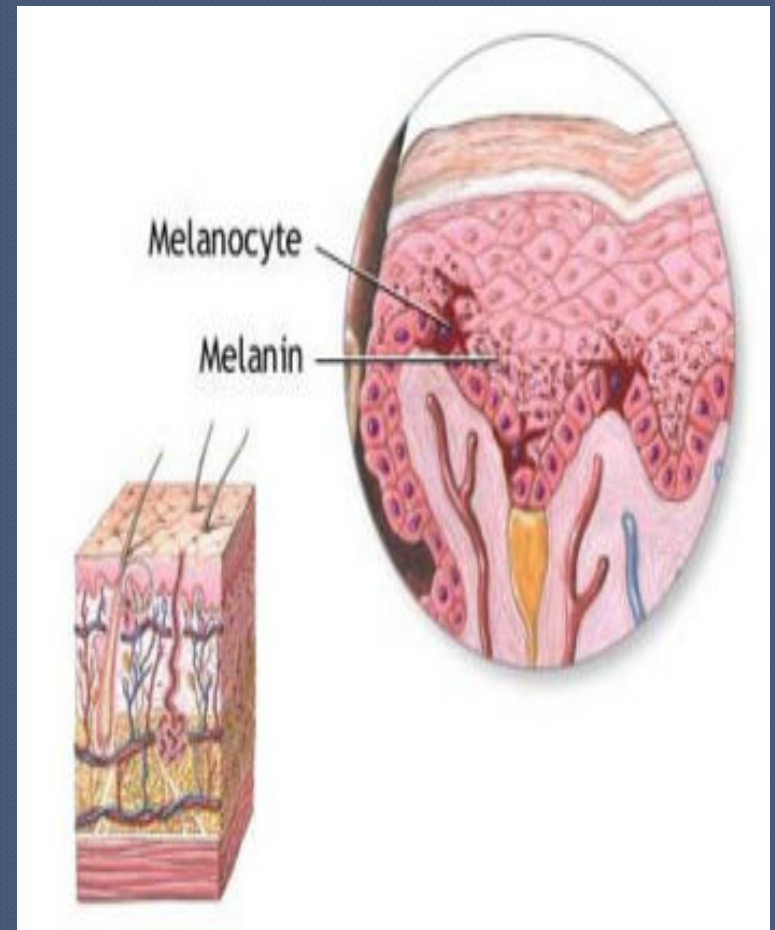
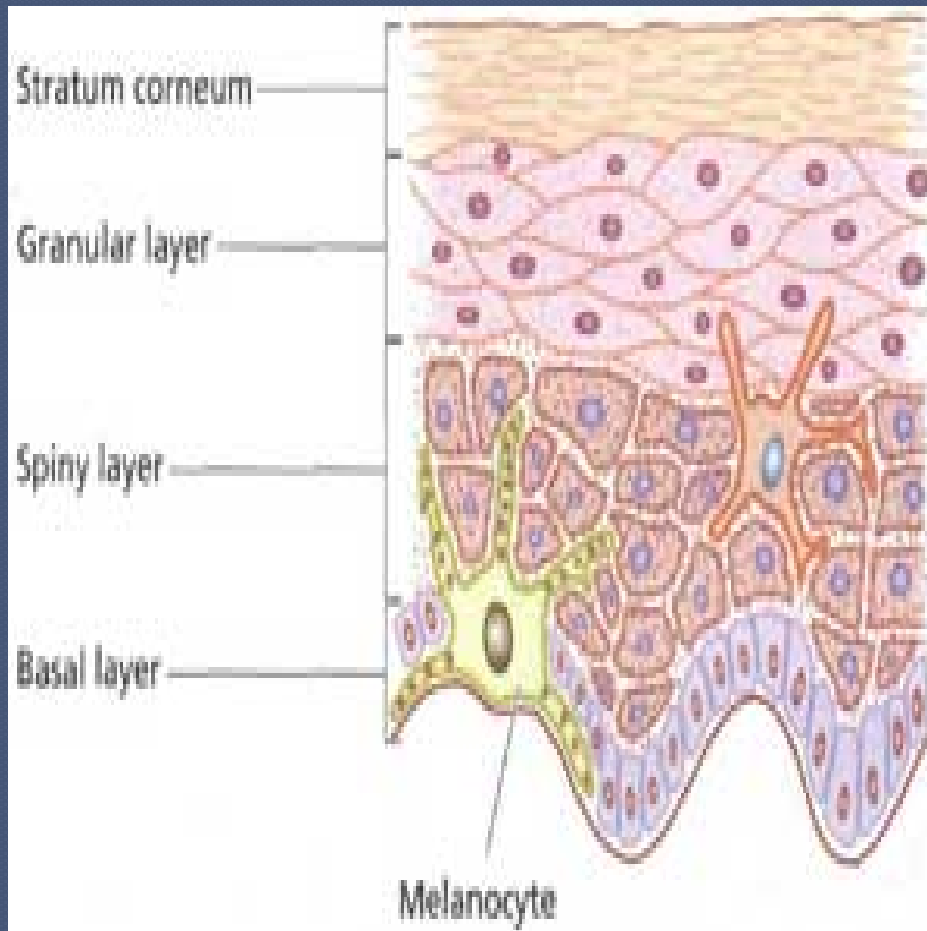
- Basal cell layer.
- Squamous cell layer,
- Granular cell layer.
- Keratinous cell layer.

Melanocytes: Formation of melanin.

Langerhan cells: Immune function of skin.

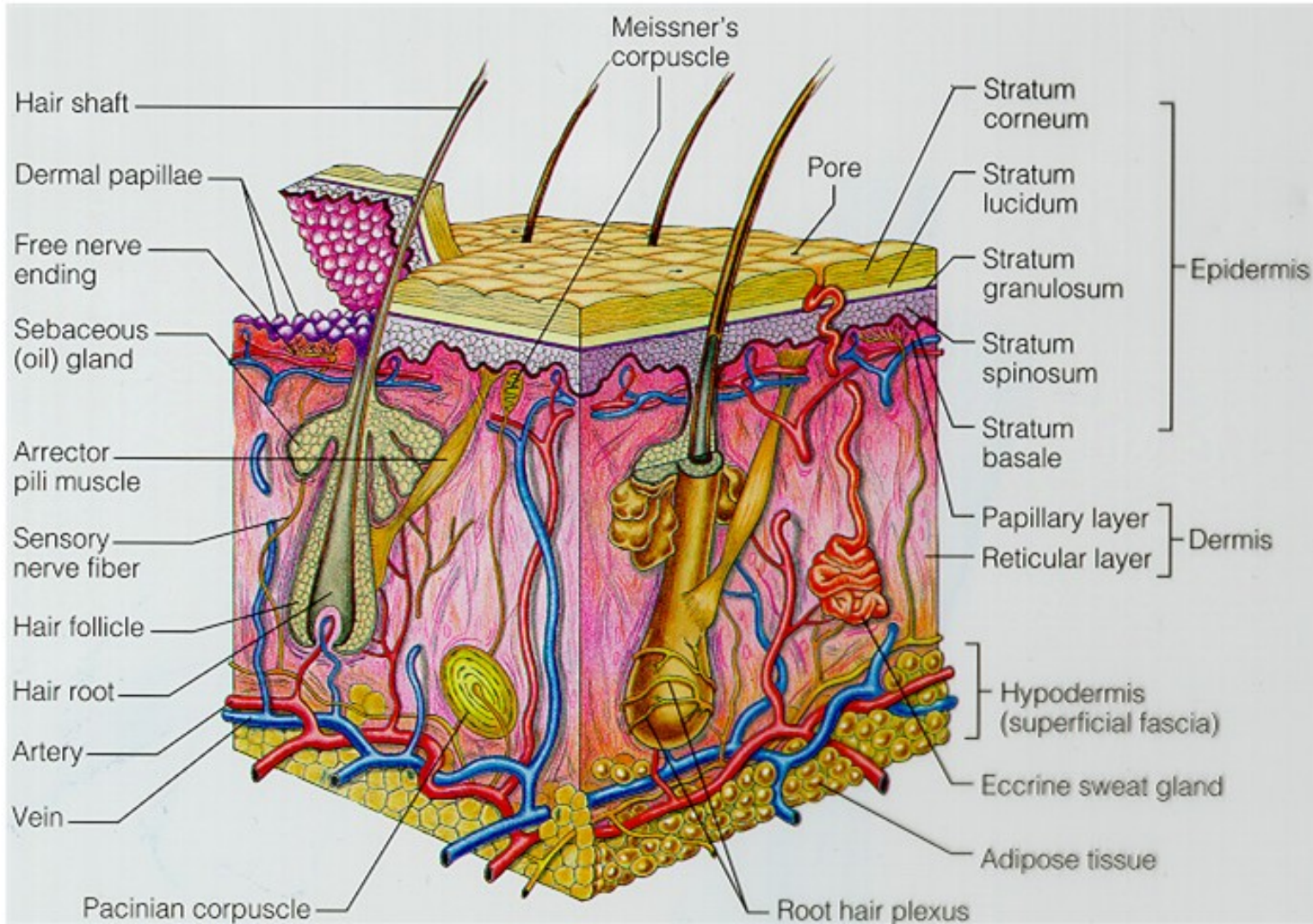
HUMAN SKIN





II. Dermis

- **Below the basement membrane.**
- **Composed of a matrix of collagen & elastic fibers.**
- **Contains**
 - **Blood vessels.**
 - **Lymphatic vessels.**
 - **Nerve fibers.**



III. Hypodermis (*SC Fat*)

Composed of lobules of fat cells,

separated by fibrous septa which are

composed of collagen & large blood vessels

Skin Appendages

a. Nail

Contain keratin.

b. Hair follicles

Also contain keratin.

c. Sebaceous glands

- Discharge their sebum content into hair follicles.
- Together with hairs, they form “**pilosebaceous units**”.

d. Sweat glands

Open on the surface of epidermis through sweat ducts

- **Eccrine sweat glands** all over body surface.
- **Apocrine sweat glands** at body flexures only.

Knowledge of normal structure of skin is important

- Any change manifests as clinical symptoms & signs.
- Any skin disease has both a visual diagnosis & a structural alteration.
- Examples
 - Absence of hair follicles manifests as **alopecia**.
 - Absence of melanocytes manifests as **vitiligo**.

Functions of Skin

1. Protection.
2. Prevention of water & electrolyte loss.
3. Temperature control through vasodilatation, vasoconstriction & sweating.
4. Sensory function.
5. Formation of vitamin D.
6. Immunological function via Langerhans cells.

Cutaneous Signs

I) Primary Lesions

Initial lesions of skin diseases (*first to appear*).

II) Secondary Lesions

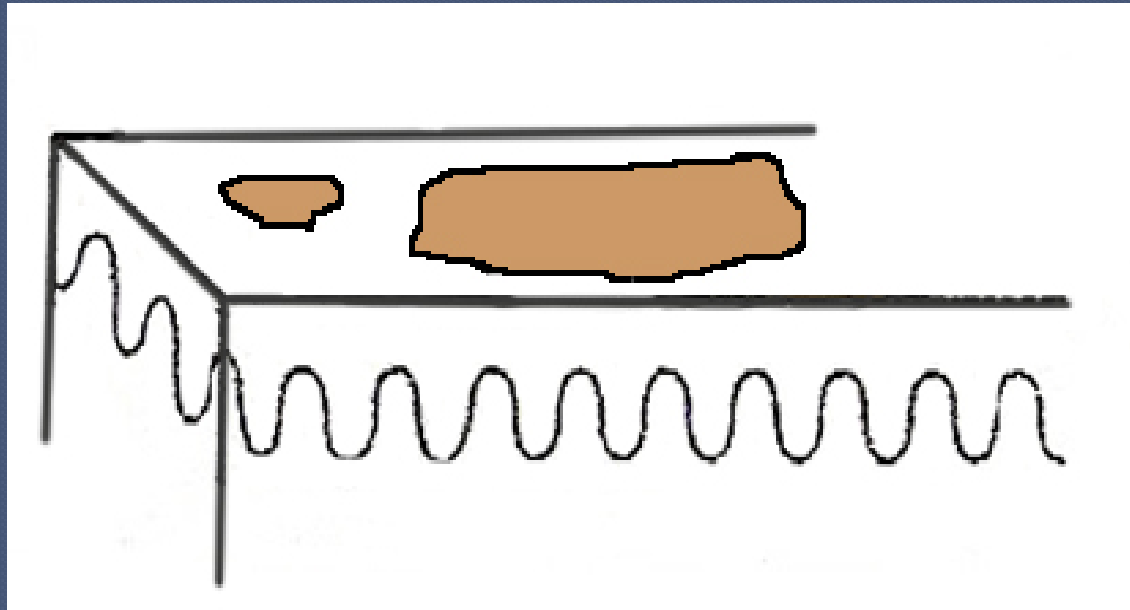
Occur as a result of modification of primary lesions.

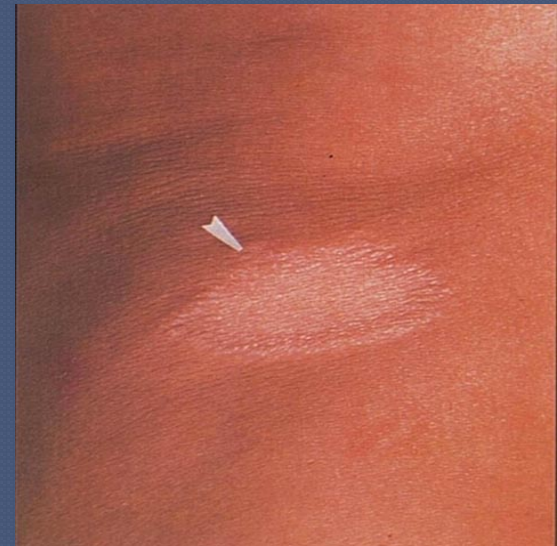
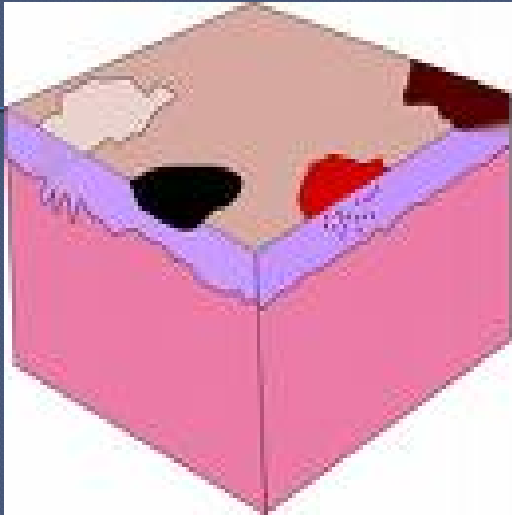
I) Primary Lesions

Macule

- Circumscribed area of skin discoloration **less than 1 cm** in diameter.
- Could be hypopigmented, hyperpigmented or erythematous.
- Examples
 - a. **Brown macule** in pityriasis versicolor.
 - b. **White macule** in vitiligo.
- A macule **more than 1 cm** in diameter is called a **patch**.

Macule & Patch



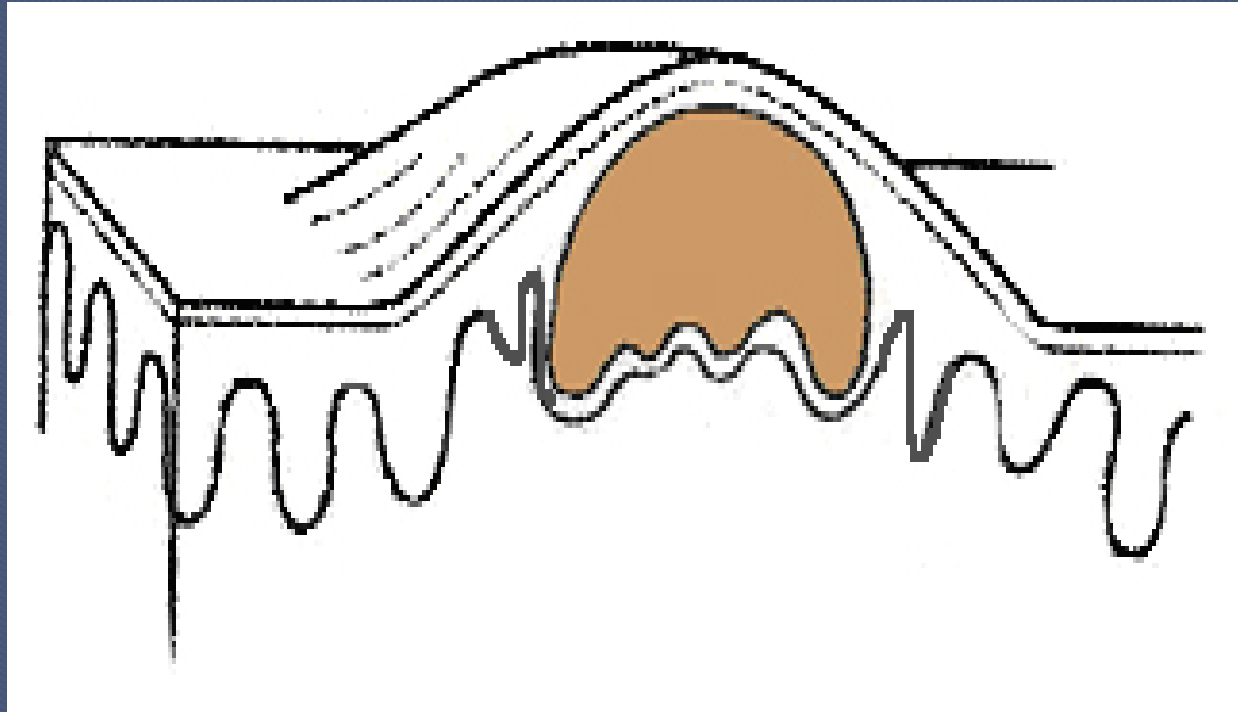




Papule

- Circumscribed solid elevation of skin **less than 0.5 cm** in diameter.
- Examples
 - a. Psoriasis.
 - b. Lichen planus.

Papule





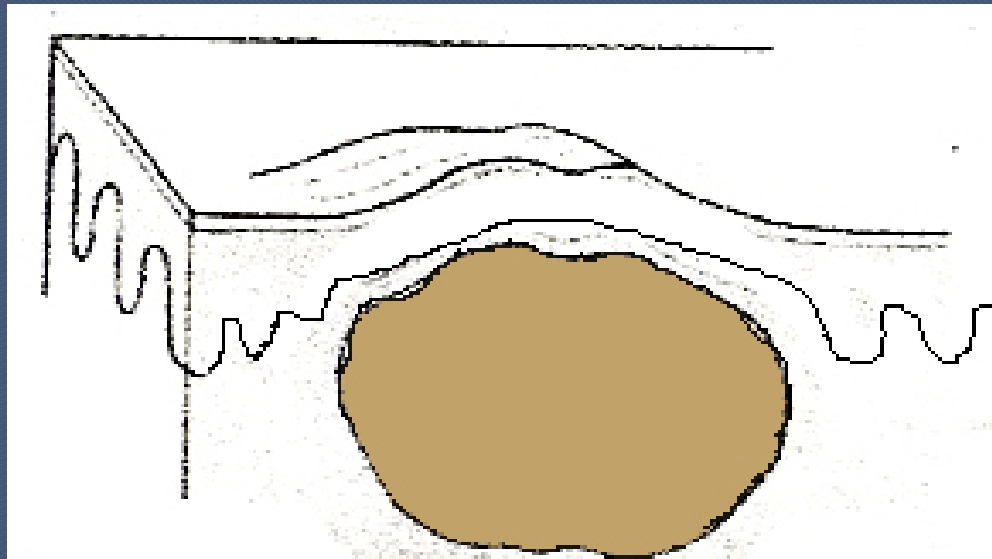
Papules



Nodule

- Circumscribed solid elevation of skin **more than 0.5 cm** in diameter.
- A deep lesion that represents a dermal or subcutaneous pathology.
- Example
 - Lepromatous leprosy.

Nodule





Plaque

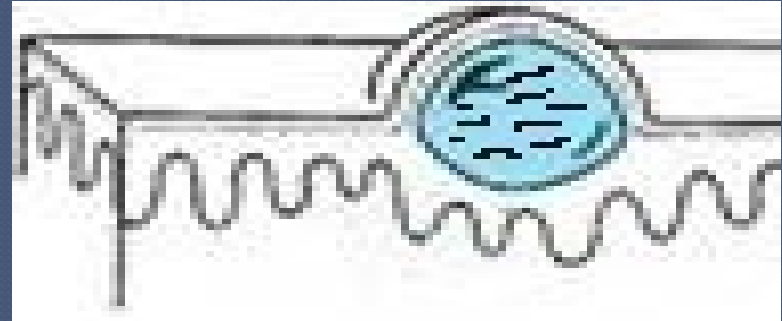
- Area of change of texture or consistency of the skin.
- May be **elevated above** or **depressed under** the skin surface.
- **An elevated lesion** may originate de novo or as a result of confluence of multiple papules.
- Occupies a large surface area in comparison with its height in contrast to the **nodule**.

Plaques



Vesicle

Elevation of skin containing fluid **less than 0.5 cm** in diameter.

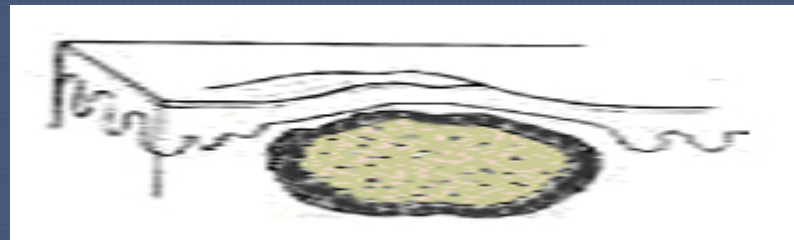


Bulla

Elevation of skin containing fluid **more than 0.5 cm** in diameter.



****A cyst** differs from a **vesicle** or a **bulla** by having a wall.



Vesicles



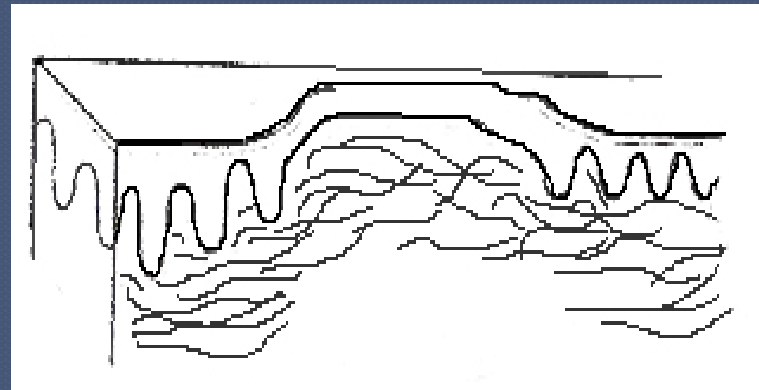
Bullae





Wheal

- Primary lesion of **urticaria**.
- Evanescent (*transient*) edematous elevations of the skin of variable sizes.
- Itching is usually present.

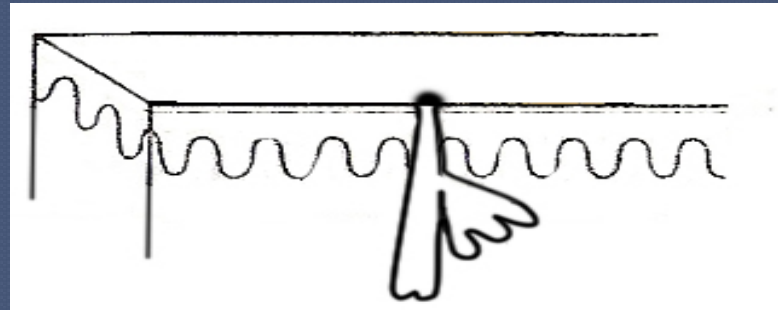


Wheals



Comedo

- Primary lesion of **acne**.
- Two types;
 - **Open comedo** or **black head**; a flat or slightly elevated papule with dilated central opening filled with blackened keratin.
 - **Closed comedo** or **white head**; a yellowish papule.

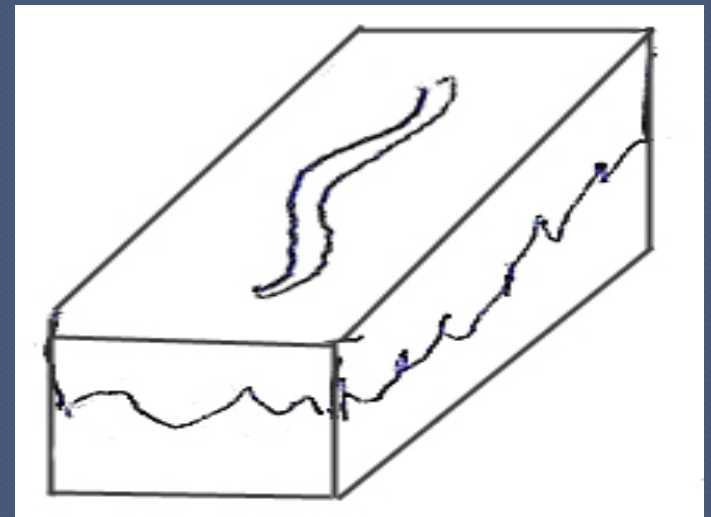
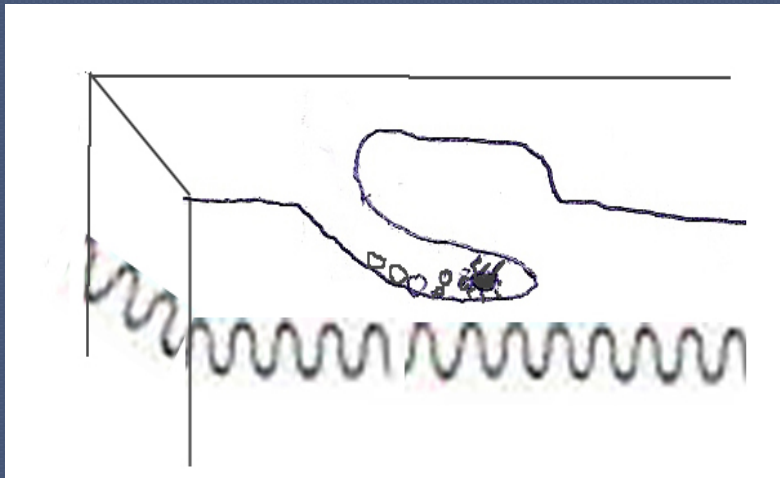


Comedones



Burrow

- Primary lesion of **scabies**.
- A linear elevation of the epidermis tunneled by the female **sarcoptes scabiei** mite.



Burrow



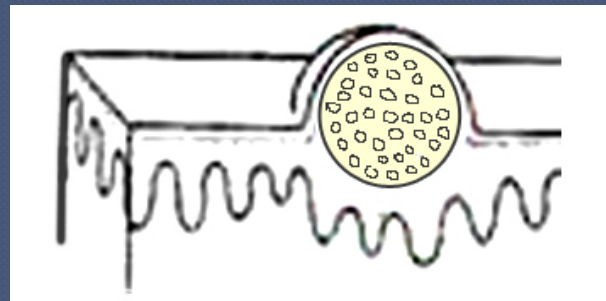
II) Secondary Lesions

Furrow

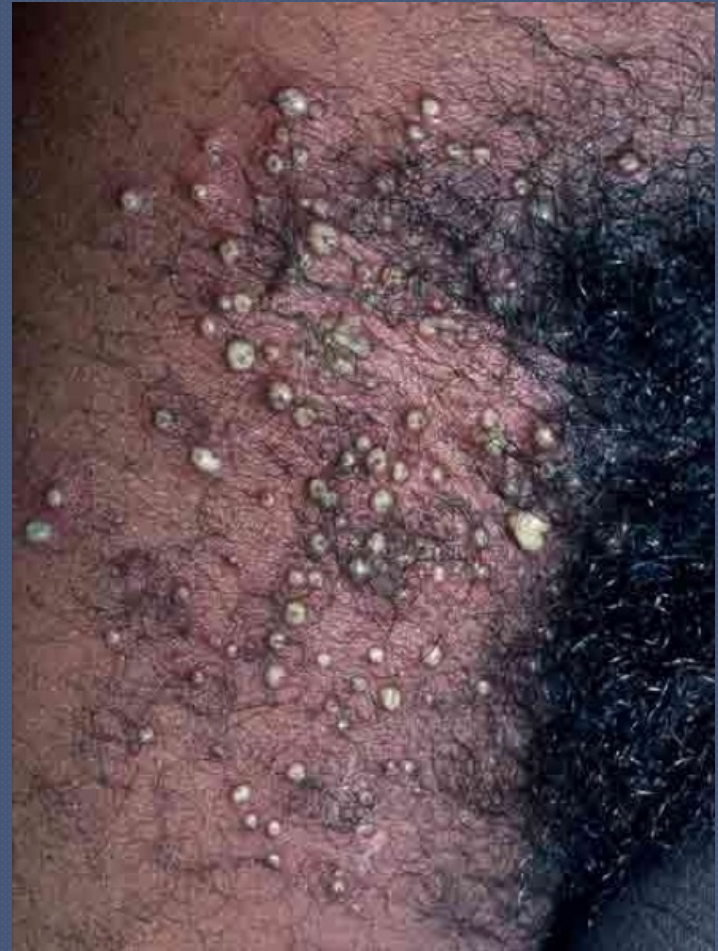
- A deroofed burrow.
- Caused by scratching of a burrow.

Pustule

- Small elevation of the skin containing purulent material.
- May originate as a pustule or may develop from a papule or vesicle (*primary or secondary*).

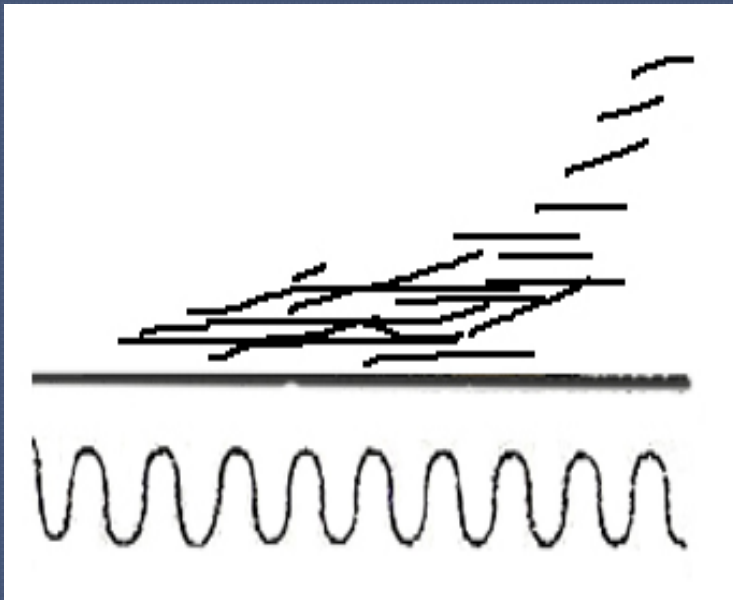


Pustules



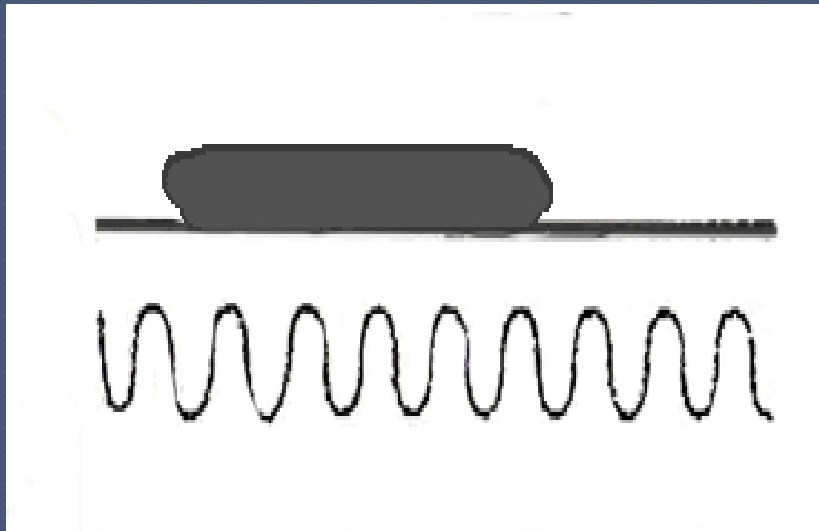
Scale

Dry **or** greasy laminated masses of keratin.



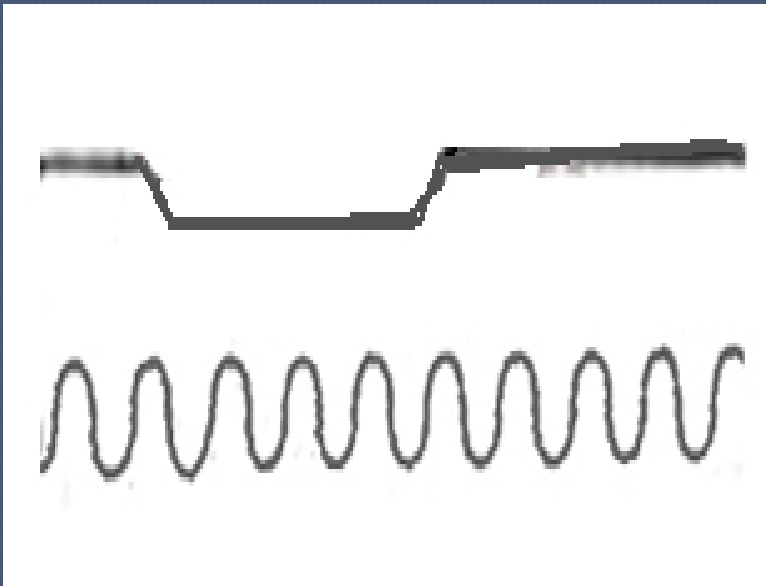
Crust

Dried material on the skin as serum, pus or blood.



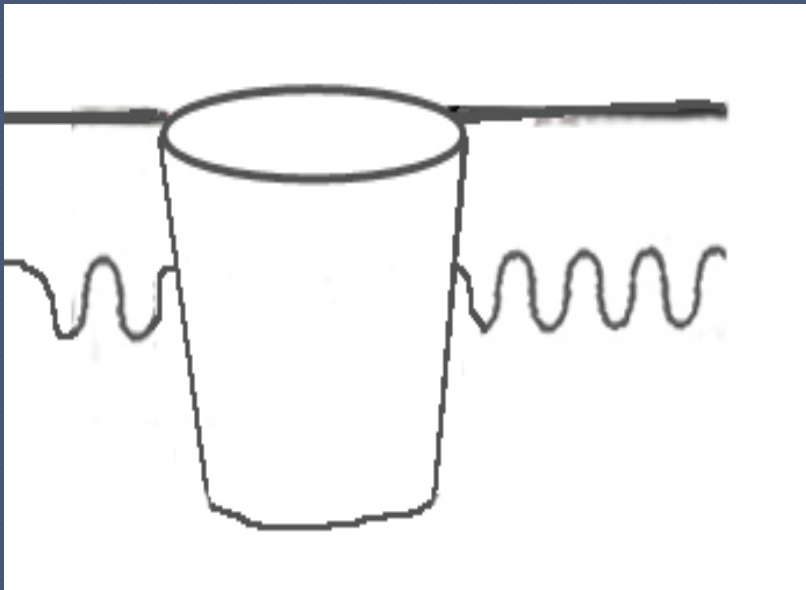
Erosion

- Partial or total loss of the epidermis, not reaching the dermis.
- Heals without a scar because the dermis is not involved.



Ulcer

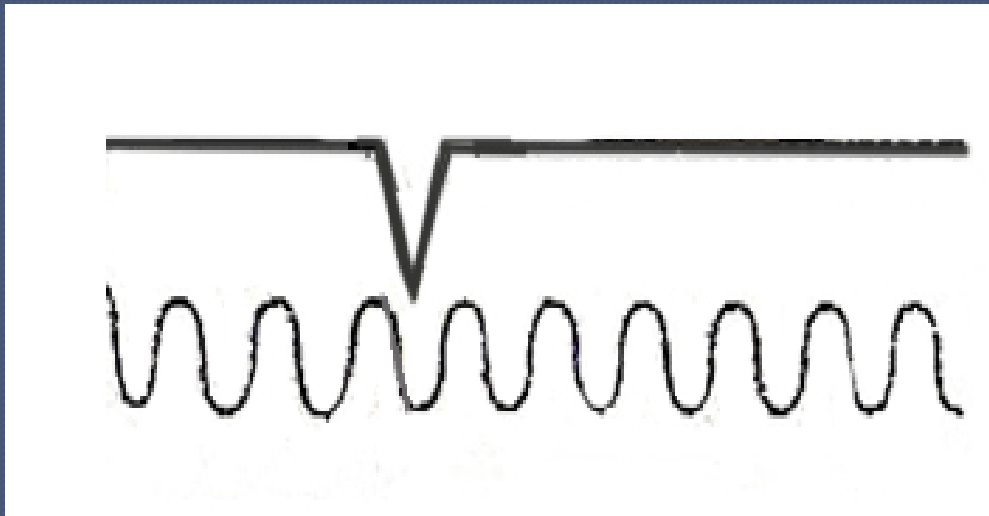
- Rounded or irregularly shaped excavations that result from total loss of the epidermis plus some portion of the dermis.
- Shape, size & depth are variable depending on disease process.



Excoriations & Abrasions

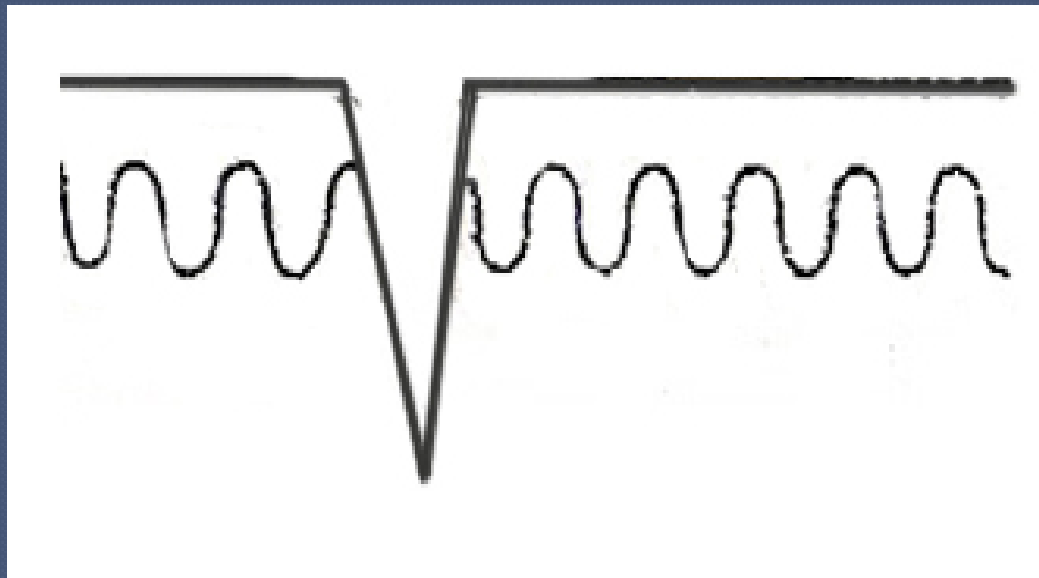
Superficial discontinuation of the skin; **only epidermal**.

- **Excoriations** are caused by scratching with fingernails.
- **Abrasions** are due to mechanical trauma or constant friction.



Fissure (Crack)

Linear cleft through **the epidermis** extending into the **dermis**.



THANK YOU

SKIN INFECTIONS

I) BACTERIAL INFECTIONS

Intended Learning Outcomes

- 1. Types & causative strains of bacterial skin infections.**
- 2. C/P of different types of bacterial skin infections.**
- 3. Complications & treatment of different types of bacterial skin infections.**

a) **Impetigo Contagiosum.**

b) **Folliculitis.**

c) **Sycosis Barbae.**

d) **Furuncle (Boil).**

e) **Carbuncle.**

f) **Erysipelas.**

g) **Cellulitis.**

h) **Erythrasma.**

a) Impetigo Contagiosum

Superficial infection of the skin

Etiology

Cocci-type bacteria; Strept. & Staph.

Infection could be

Primary due to poor hygiene & moisture.

Secondary due to insect bites, scabies or pediculosis capitis infestation.

Clinical Types

1. Ordinary Impetigo.
2. Bullous Impetigo.
3. Circinate Impetigo.
4. Ulcerative Impetigo (Ecthyma).

1. Ordinary Impetigo

- **Vesicles** → Rupture → Seropurulent discharge → Dries → Golden yellow crusts.
- On face, hands, genitalia & scalp (*pediculosis capitis infestation*).
- No constitutional symptoms.
- Resolves within a few days.



Ordinary Impetigo





2. Bullous Impetigo

- **Staphylococci.**
- Primary lesion is a **bulla.**
- Newborn infants or any age.
- Accompanied by constitutional symptoms.
- Might be fatal in newborn infants.

3. Circinate Impetigo

- Extension of ordinary impetigo or secondary to rupture of bullous impetigo.
- Lesions are **circinate**.



4. Ulcerative Impetigo (Ecthyma)

- Occurs on **legs**.
- Lesions have **thick crusts**.
- Heals with **scars**.



Complications

1. Spread of infection **to other sites.**
2. Spread of infection **to other children.**
3. **Post streptococcal glomerulonephritis** in 2-5% of cases.

Treatment

1. **Removal of crusts.**
2. **Topical antiseptics** as povidone iodine or KMnO_4 1/8000-1/10,000.
3. **Topical antibiotics** as fusidic acid, gentamycin or bacitracin cream.
4. **Systemic antibiotics** given if infection is generalized, associated with fever or lymphadenopathy or in cases of bullous impetigo or ecthyma.

b) Folliculitis

Follicular infection in upper part of hair follicle

Etiology

Staphylococcus aureus.

Predisposing factors

Moisture & poor hygiene.

C/P

Follicular pustules.

Treatment

1. Treatment of predisposing factors.
2. Topical antiseptics.
3. Topical & systemic antibiotics.

Folliculitis



Scalp Folliculitis



c) Sycosis Barbae

Folliculitis of the beard area

Etiology

Staphylococcus aureus.

Predisposing factors

Moisture, poor hygiene & shaving.

C/P

Follicular pustules & papules in the beard area.

Treatment

1. Treatment of predisposing factors.
2. Topical antiseptics.
3. Topical & systemic antibiotics.

Sycosis Barbae



d) Furuncle (Boil)

**Deep infection in lower part of hair follicle
with central necrosis**

Predisposing factors

Obesity & diabetes mellitus.

C/P

Follicular red **papules**.

Treatment

1. Treatment of predisposing factors.
2. Topical antiseptics.
3. Topical & systemic antibiotics.

Furuncle



e) Carbuncle

Multiple deep boils that open on the surface
by multiple fistulae

Predisposing Factor

Diabetes.

C/P

- **Multiple deep boils that open on the surface by multiple fistulae.**
- **On the back, neck & intertriginous areas.**

Treatment

- 1. Incision & drainage.**
- 2. Systemic antibiotics.**

Carbuncle



f) Erysipelas

Infection of upper dermis

Etiology

Beta-hemolytic Streptococci.

Predisposing factor

Lymphedema.

C/P

Erythematous, swollen, tender area with a **sharp border** affecting frequently legs & face. In extensive cases, blisters may be formed. Constitutional symptoms include malaise & fever.

Complications

Lymphedema occurs from recurrent episodes.

Treatment

Systemic antibiotics as penicillin or erythromycin.

Erysipelas



g) Cellulitis

Suppurative infection of lower dermis & SC tissue

Etiology

Staphylococcus aureus & Streptococcus pyogenes.

C/P

- Erythematous, swollen, tender area with an **ill-defined border**.
- **Constitutional symptoms** include malaise, chills & fever.

Treatment

Aggressive antibiotic therapy

Systemic antibiotics as penicillin or erythromycin.

h) Erythrasma

Superficial infection of intertriginous areas

Etiology

Corynebacterium minutissimum.

Predisposing factors

Obesity, diabetes & debilitating diseases.

C/P

- Dry, scaly, reddish-brown **patches** with fine scales in **intertriginous areas**; axillae, groins & submammary areas.
- **Wood's light** → **Coral red fluorescence**.

DD: Tinea cruris, flexural candidiasis & flexural psoriasis.

Treatment

I) Topical Therapy

- **Antibiotics** as fusidic acid. · **Antifungals** as azoles.

II) Systemic Therapy

- **Antibiotics** as erythromycin or tetracycline.

Erythrasma





THANK YOU

V) LEPROSY
(HANSEN'S DISEASE)
(HANSENIASIS)

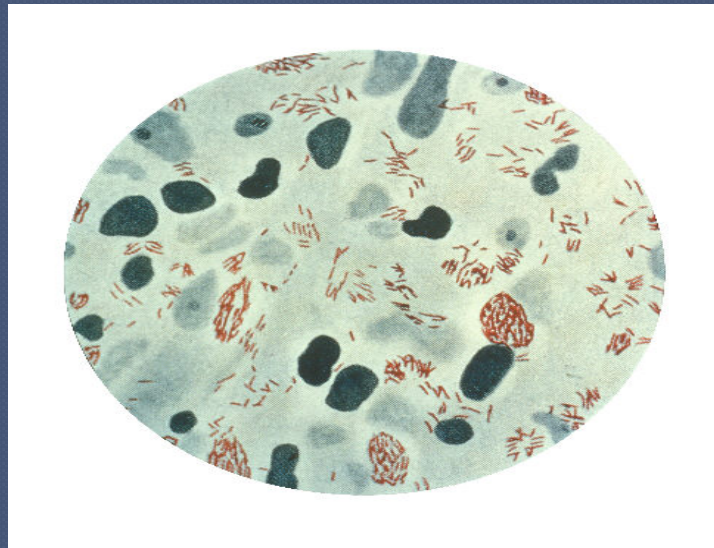
Intended Learning Outcomes

- 1. Etiology & mode of infection of leprosy.**
- 2. Classification & immunity of leprotics.**
- 3. Diagnosis of different types.**
- 4. Reactions in leprosy.**
- 5. Treatment guidelines of leprosy.**

-
- **Chronic** granulomatous mycobacterial infectious disease.
 - **Occurs more in** tropical & subtropical areas.
 - **Targets** mainly skin & nerves.
 - **Incubation period** is long ranging from 2-12 years.

Mycobacterium leprae

- Slightly curved bacilli.
- Acid & alcohol fast.
- Stained by **modified Ziehl–Neelson stain**.
- Do not grow in vitro but grow in **footpads of laboratory animals**.



Pathogenesis of Infection

- Route of Infection: Nasal mucosa or wounds.

Cell-mediated immunity

- Bacilli enter through nasal mucosa → Engulfed by Schwann cells → Clinical manifestations according to immune status of the body in a wide range of spectrum!

TT ↔ BB ↔ LL

- Immune status of the host

- **High immunity** → Paucibacillary leprosy.
- **Poor immunity** → Multibacillary leprosy.

Classification of leprosy

I) Bacteriological classification

a. Paucibacillary leprosy

- Tuberculoid leprosy (TT).

b. Multibacillary leprosy

- Borderline leprosy (BB).
- Lepromatous leprosy (LL).

Classification of leprosy (cont.)

II) Clinical classification

- a. Tuberculoid Leprosy (TT).
- b. Borderline Leprosy (BL).
- c. Lepromatous Leprosy (LL).

Nerve Involvement in Leprosy

- Superficial nerves

- Ulnar, lateral popliteal & great auricular nerves.
- Involved nerves are thickened, beaded & tender.

- Sensory nerves affection

Glove & stock anesthesia → Loss of pain → Repeated injuries of hands & feet → Trophic ulcers.

- Motor nerves affection

- Facial nerve → Facial palsy.
- Ulnar nerve → Claw hand.
- Median nerve → Ape hand.
- Lateral popliteal → Dropped foot.

	1) Tuberculoid Leprosy	2) Lepromatous Leprosy
Disease of	Nerves & skin.	Nerves, skin & systemic.
Immunity	Good.	No Immunity.
Bacteriology	Paucibacillary.	Multibacillary.
C/P	<ul style="list-style-type: none"> • Maculoanaesthetic patch with hypopigmented centre. • Skin is dry, hairless & insensitive. 	<ul style="list-style-type: none"> • Glistening erythematous or skin-colored papules, nodules & plaques. • Ears → Thickened & nodular. ** Thickening of ears, face & skin of forehead + Nodules on nose + Deepening of natural lines gives the picture of "leonine facies". • Eye brows → Alopecia of outer 1/3. • Legs → Edema & ulceration.

	1) Tuberculoid Leprosy	2) Lepromatous Leprosy
Nerves	Involved early → Glove & stocking anesthesia.	Late anesthesia → Loss of temperature, light touch, pain then deep touch → Trophic ulcers.
Mucous Membranes	None.	<u>Nose</u> <ul style="list-style-type: none"> • Spontaneous bleeding. • Nodules & ulcers of septum. • Cartilage destruction & deformity.
Others		<ul style="list-style-type: none"> • Larynx, bones, muscles & testes. • Intercurrent infections.

Tuberculoid Leprosy





Untreated chronic TT with peripheral neuropathy



LL nodules



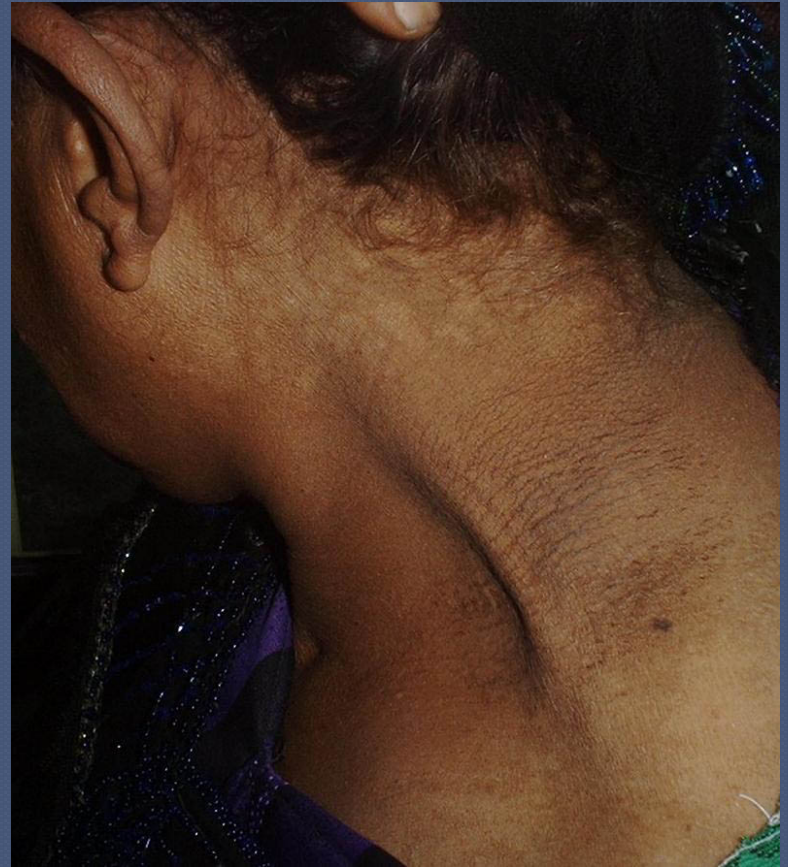
Lepromatous Leprosy



LL with stigmata



Borderline Leprosy







Diagnosis

1. **Clinical picture.**
2. **Skin smear** from ear lobes, elbows, knees & any visible lesion, then stained with **modified Ziehl–Neelson stain.**
3. **Skin biopsy.**
4. **Nerve sheath biopsy.**
5. **Polymerase chain reaction.**

Reactions in Leprosy

- Acute episodes that occur during the chronic course of multibacillary leprosy.
- May occur
spontaneously *or*
precipitated by treatment, infections, physical stress, injury, operations, pregnancy, parturition or vaccination.

Course of Leprosy: Very slow course.

Treatment of Leprosy

I) General Lines

- Health care.
- Patient education & rehabilitation.

II) Chemotherapy

- **Rifampicin** +
- **Dapsone** → Cheapest & most important drug.
- **Clofazimine** → Bacteriostatic & anti-inflammatory.

THANK YOU

II) VIRAL INFECTIONS

Intended Learning Outcomes

- 1. Most common viral skin infections & their causative viruses.**
- 2. C/P of different types of viral skin infections.**
- 3. Differentiation between herpes simplex & herpes zoster.**
- 4. Treatment of different types of viral skin infections.**

Most Important viral skin infections include

- a) **Herpes simplex → Herpes simplex virus (HSV).**
- b) **Herpes Zoster → Varicella zoster virus (VZV).**
- c) **Warts (Verrucae) → Human papilloma virus (HPV).**
- d) **Molluscum Contagiosum → Pox virus.**

a) Herpes Simplex (HS)

Most common viral infection

- Herpes simplex virus (HSV).
- Two types
 - HSV type I → Non-genital infections.
 - HSV type II → Herpes progenitalis.
- Mode of Transmission
 - Skin-to-skin.
 - Skin-to-mucous membrane contact.

HSV Type I

Primary Infection *Primary Herpetic Gingivostomatitis*

- Individuals infected for the first time.
- Subclinical in about 90% of cases.
- Small superficial **vesicles** on the oropharynx → Rupture quickly → Painful denuded areas.
- Swollen gums, fever, sore throat, malaise, loss of appetite & lymphadenopathy.
- Lesions heal within 2 weeks.

Primary HS



Recurrent Attacks

- Following resolution of primary infection →
Neural tissue (*dorsal root ganglia*) → Remains
dormant → If reactivated, viral particles migrate
along peripheral nerves to skin & mucous membranes →
Recurrent HS at or near primary site
- Predisposing Factors for Reactivation
Fever, fatigue, trauma, UV radiation, stress,
menstruation, GIT disturbances, altered immune status
or immunosuppressives.

- Sites of Recurrent Attacks

Lips

Herpes Labialis

Bilateral grouped **vesicles** on erythematous base on the lips usually preceded or associated with a burning or tingling sensation → Within a few days, **vesicles** dry up forming crusts and fall off → Lesions heal without a scar

Face

Herpes Facialis

Around other orifices as nose, eyes, ears & cheeks.

Recurrent HS





Special Variants of HSV Type I

- Ocular Mucosa

- Ophthalmologic consultation; may lead to corneal opacity.

- Finger or Hand

Herpetic Whitlow

- On the finger as a result of direct inoculation.
- Very painful with **vesicles**, edema & redness.

Periocular HS



Herpetic whitlow



One or more small tender vesicles, typically on the distal phalanx, characterize herpes simplex infection of the fingers.

Herpes Simplex Type II

Primary Infection

Herpes progenitalis

- Transmitted by sexual contact.
- Involves genitalia of both males & females.
- Painful grouped superficial vesicles on an erythematous base → Erosion or genital ulceration.
- Herpes progenitalis in a pregnant woman at the time of delivery → **Caesarian section.**

Recurrent Attacks

- Less severe than the primary infection.

Genital HS



Complications of HSV Infection

1. **Secondary infection.**
2. **Eye complications;** keratitis & corneal ulcers.
3. **CNS complications;** encephalitis & meningitis.
4. **Erythema multiforme** due to recurrent HSV.
5. **Cancer cervix** due to recurrent cervical HSV.

Diagnosis of HS

1. C/P.
2. Tzank smear.
3. Viral culture.
4. Serological tests.
5. PCR.

Treatment of HS

- I. **Avoid** precipitating factors.
- II. **Avoid** direct sexual contact during the attack in genital herpes.
- III. **Topical Therapy** *In vesicular stage*
 - **Drying antiseptic lotions;** KMnO_4 1/8000-1/10,000 - 10% aluminum acetate.
 - **Antivirals (acyclovir cream)** 5 times daily for 5 days.
 - **Idoxuridine (IDU)** for eye lesions.
- IV. **Systemic Therapy**
 - **Acyclovir**, 200 mg 5 times daily for 5 days.

b) Herpes Zoster (HZ)

- **Varicella zoster virus (VZV).**
- **Chicken Pox (Varicella)**
 - Patient exposed to the virus for the first time.
 - Childhood disease with generalized self-limiting **vesicular** eruption → Brownish crust → Lesions heal within 10 days → After attack, virus resides in posterior root ganglia

Varicella





Reactivation of Infection (Herpes Zoster)

- Predisposed to by

Trauma, fever, decreased resistance, drugs, diseases of spine & malignancy.

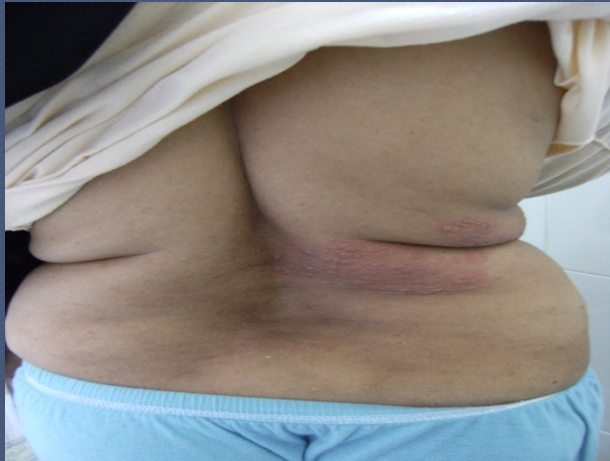
- C/P

- Onset is accompanied or preceded by pain → Unilateral groups of **vesicles** on erythematous & edematous base, along distribution of one or more sensory nerves → Local LNs may be enlarged → **Vesicles** dry up without rupture → Recovery occurs after 2-4 weeks → May leave scars.
- Lesions may be **hemorrhagic, gangrenous, generalized** or **abortive**.
- One attack gives **permanent immunity**.

Intercostal HZ



Zoster





HZ of lower limbs



HZ Facialis

FIGURE 2. Case of herpes zoster ophthalmicus



Photo/MN Oxman, University of California, San Diego



Complications of HZ

1. **Secondary infection.**
2. **Eye complications; HZ ophthalmicus.**
3. **CNS complications; post-herpetic neuralgia.**

Superinfected HZ



Disseminated HZ



****HZ may be a manifestation of internal malignancy if**

- 1. Very old age.**
- 2. Gangrenous type.**
- 3. Bilateral affection.**
- 4. Recurrent.**

Treatment of HZ

I. Topical Therapy

In vesicular stage

- **Antiseptic drying lotions.**
- **Acyclovir cream** in very early vesicular stage, 5 times daily may minimize duration of the attack.

II. Systemic Therapy

- **Antivirals**
 - **Acyclovir**, 800 mg 5 times daily (every 4 hrs) for 7 days.
 - **Valacyclovir**, 1000 mg 3 times daily for 7 days.
 - **Famcyclovir**, 500 mg 3 times daily for 7 days.
- **Analgesics, carbamazepine & gabapentin** for pain.

c) Warts (Verrucae)

- Common, infectious, benign, epithelial growths.
- Human papilloma virus (HPV).
- Involve both skin & mucous membranes.
- Mode of Infection
Direct or indirect contact.
- Incubation Period
1-6 months.

Types of Warts

1. Common warts

Verruca Vulgaris

Asymptomatic skin-colored & verrucous papules.





2. Plane warts

Verruca Plana

- Asymptomatic skin-colored, flat-topped, slightly elevated **papules**.
- Occurs more in children.
- Shows Koebner's phenomenon.

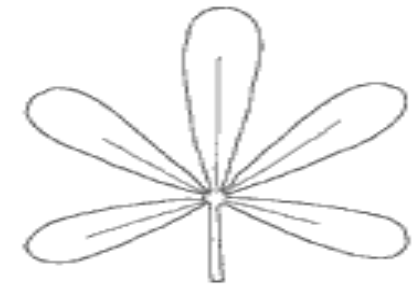


3. Filiform warts

Verruca Filiformis

Long, thin, pedunculated skin growths.





digitate

4. Digitiform warts *Verruca Digitata*

Papillomatous thin projections with finger-like processes, having a common stem.



5. Plantar warts

Verruca Plantaris

Involves sole of foot, **tender**, thick & growing inwards.



6. Genital warts *Condyloma Acuminata*

- Involves **skin** of genitals in both sexes.
- In **mucous membranes**, they are soft, pinkish, moist, foul-smelling outgrowths that bleed easily with a cauliflower appearance.



Complication of HPV Infection

Oncogenicity; cervical dysplasia or cancer cervix.

Course of Infection

- Spontaneous involution may occur within 2 years.
- Treatment of a few warts may induce regression of other warts.

Treatment of Warts

1. **Electrocautery**; cell destruction by heat effect.
2. **Cryotherapy**; cell destruction by freezing effect of liquid nitrogen.
3. **Chemical cautery**; cell destruction by caustics.
4. **Laser treatment** by CO₂ laser or pulsed dye laser.

Treatment of Warts (cont.)

5. Podophyllin resin 25%

- In alcohol, liquid paraffin or tincture benzoin co.
- Effective in treating venereal warts.
- Used as paint twice weekly.
- Should be washed after 6-8 hrs.
- Contraindicated in pregnant & in large bleeding warts.

***Imiquimod cream may be used in ttt of venereal warts by stimulating local interferon production.*

6. Autosuggestion.

7. Radiotherapy for resistant plantar warts.

d) Molluscum Contagiosum

- **Pox virus.**
- Mode of Infection: Direct or indirect contact.
- Incubation period: 2-6 weeks.
- C/P
 - Shiny, pearly white, dome-shaped **papules** with a smooth surface & central umbilication.
 - A white cheesy material can be expressed from the central punctum on squeezing the lesion.
 - Involves non-genital skin or genital skin (STD).

- **Treatment**

1. **Electrocautery.**
2. **Cryotherapy.**
3. **Chemical cautery with phenol.**
4. **Laser treatment.**





THANK YOU

IV) PITYRIASIS ROSEA (PR)

Intended Learning Outcomes

- Identification & diagnosis of pityriasis rosea.

-
- Inflammatory non-infectious scaly erythematous eruption.
 - Exanthematous reaction to an upper respiratory viral infection.
 - Highest between 15-40 years.
 - More prevalent in spring & autumn.

Etiology

Human herpes virus (HHV)-6 & -7.

C/P

Primary Lesion

Herald patch

- Single oval lesion with three different zones.
- Starts on one side of the trunk with its longitudinal axis parallel to ribs.

C/P (cont.) Secondary Eruption

- Occurs after 1-2 weeks from the onset of **herald patch**.
- Similar to **herald patch**, but smaller & multiple.
- Distributed along long axis of ribs
(Christmas tree pattern).
- Located on the trunk & proximal parts of the limbs;
flannel area giving picture of **jacket with short sleeves**.
- Itching.
- Spontaneously heal within 4-8 weeks.
- Recurrences are not common.

Herald Patch





Secondary Eruption











Differential Diagnosis

Tinea circinata (*by CP & scraping*).

Treatment

1. Patient reassurance.
2. Avoid skin irritation.
3. Soothing lotions, e.g. calamine lotion.
4. Oral antihistamines, topical corticosteroids & UVB.

THANK YOU

III) FUNGAL INFECTIONS

Intended Learning Outcomes

1. Dermatophytic infections & their causative fungi.
2. C/P of the most common types of dermatophytic infections.
3. Management of different types of dermatophytic infections.
4. Yeast infections & the nature of their causative organisms.
5. Cause, C/P & management of pityriasis versicolor.
6. Cause, C/P & management of mucocutaneous candidiasis.
7. Antifungals & their indications.

Fungal skin diseases

are either

Superficial → Skin only

or

Deep → Internal organs

SUPERFICIAL FUNGAL INFECTIONS

Among the most common dermatologic disorders

Superficial Fungal Infections

a. Dermatophytes

b. Yeasts

- Malassezia furfur.
- Candida species.

DERMATOPHYTES

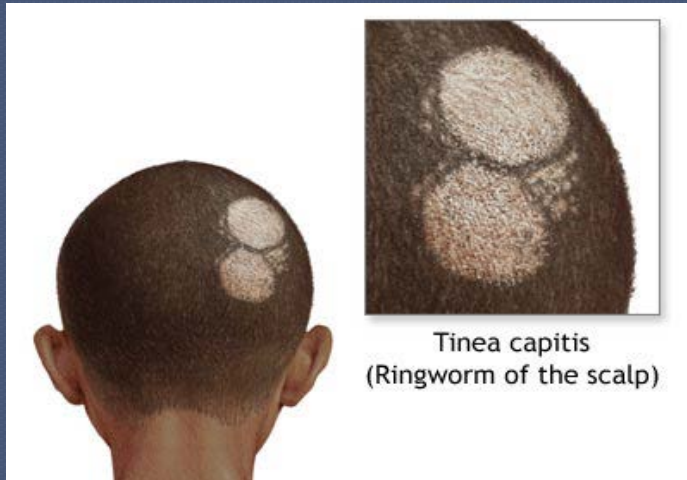
- Known as **ringworm** or **tinea**.
- Involve skin & skin appendages (*hair & nails*).
- Mode of Infection: Direct or indirect.
- Clinical Classification
 1. **Tinea Capitis** → Ringworm of **scalp**.
 2. **Tinea Corporis (Circinata)** → Ringworm of **trunk**.
 3. **Tinea Pedis** → Ringworm of **feet**.
 4. **Onychomycosis** → Fungus of **nails**.

1. Tinea Capitis

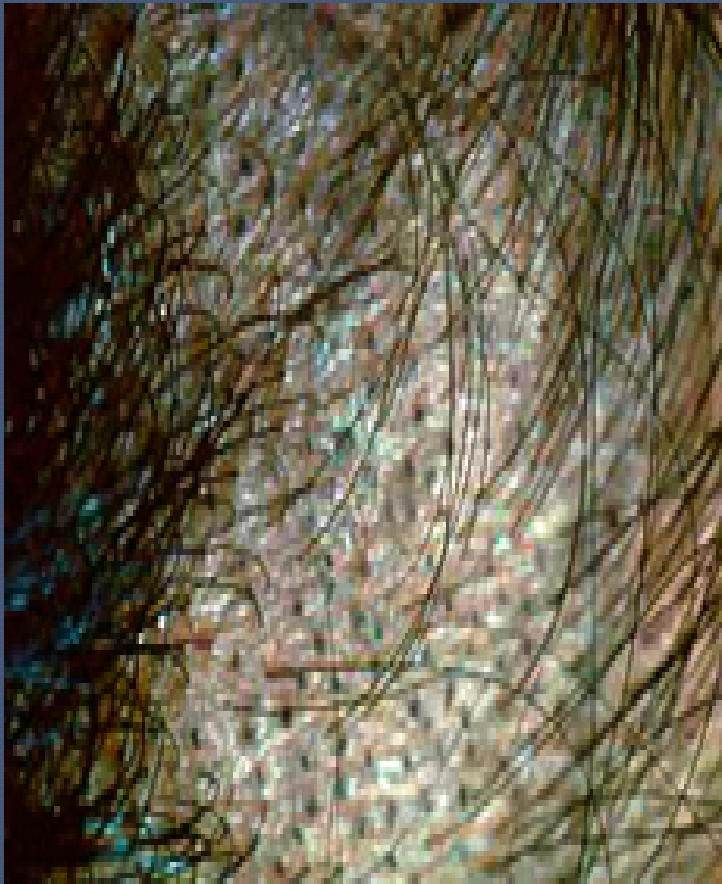
	a. Scaly Type	b. Black-Dot Type	c. Kerion	d. Favus
Epidemiology	Children only.	Children only.	Children & adults.	Children & adults.
C/P	Single or multiple bald patches on the scalp, with fine grayish-white scales. Loose hairs that break off → Stumps that can be easily pulled out.	Hairs break off at the surface of the skin giving picture of black dots.	Boggy swelling studded with follicular pustules, pus is localized to hair follicles.	Yellow cup-shaped sulfur crusts (<i>scutula</i>) of mousy odor that form around loose hairs and lead to a diffuse loss of hair replaced by fibrous tissue.

	a. Scaly Type	b. Black-Dot Type	c. Kerion	d. Favus
DD	Alopecia areata.		Abscess.	
Course	No scar.	No scar.	Cicatricial alopecia.	Cicatricial alopecia.

Scaly Ringworm of Scalp



Black Dot Ringworm



Favus



Diagnosis

1. **Clinical examination.**
2. **Fluorescence** under wood's light.
3. **Direct microscopic examination.**
4. **Culture.**

Treatment of tinea capitis

Mainly systemic

Systemic Therapy

Griseofulvin, 12.5 mg/kg/day (*one tablet/10 kg body wt. with a maximum of 6 tablets*) for 6-8 weeks.

Topical Therapy

used to decrease infectivity; useless if used alone

- **Tincture iodine 1-2%.**
- **Whitfield ointment.**
- **Broad spectrum antifungals.**
- **Ketoconazole shampoo.**

2. Tinea Circinata

Tinea Corporis

C/P

- Single or multiple well-defined annular patches.
- Sometimes lesions coalesce to form polycyclic patterns.
- Occurs on the body and given the name according to the affected area e.g. **tinea axillaris**, **tinea cruris**, **tinea barbae** & **tinea mannum**.
- Itching.

Differential diagnosis

Herald patch of pityriasis rosea.

Treatment of Tinea circinata

- I. **For localized cases**, topical antifungal, twice daily.
- II. **For extensive or resistant cases**, oral **griseofulvin** for 3 weeks, **azole derivatives** or **allylamines**.

Tinea Circinata







****Tinea of flexural areas (axillaris, cruris)**

Predisposing Factors

Heat, friction, obesity, excessive sweating & maceration.

Differential diagnosis

1. **Candidiasis.**
2. **Flexural psoriasis.**
3. **Erythrasma.**

Tinea Cruris



****Tinea Barbae**

Source of Infection

Farm animals or barber's instruments.

C/P

May be *T. circinata*-like or *kerion*-like.

Tinea Barbae



3. Tinea Pedis

Athlete's Foot

Predisposing Factors

Excessive sweating & excessive moisture.

Source of Infection

Wet floor boards.

C/P

- Skin between toes, particularly the 4th & 5th is sodden, white & macerated with a bad odor.
- Mostly bilateral.
- Recurrences are common.

Tinea Pedis



Image Courtesy of C. Halde
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4. Tinea Ungium

Onychomycosis

C/P

- Yellowish or greenish coloration with thickening & brittling → Onycholysis.
- One or more nails of digits or toes may be involved, usually unilateral & asymmetrical if bilateral.

Treatment

Systemic.

Onychomycosis





YEASTS

Malassezia furfur → Pityriasis Versicolor

Candida species → Candidiasis (Moniliasis)

a) Pityriasis Versicolor

- A very common superficial mycotic infection.
- More in tropical climates & summer time.
- Occurs in young adults with a familial predisposition.
- Caused by lipophilic yeast **Malassezia furfur**; the pathogenic (mycelial) form of **Pityrosporum orbiculare**.

C/P

- Sharply demarcated **macule**, hyperpigmented or hypopigmented covered by **fine branny scaling**.
- Usually starts on neck, upper parts of chest, back of arms. In extensive cases, it spreads to abdomen & other parts of body.
- Symptomless, but itching may be present.
- Recurrences are common in summer due to heat & humidity.
- Untreated cases may persist for years.

Pityriasis Versicolor



Diagnosis

1. **Clinical examination.**
2. **Wood's light** gives yellow fluorescence.
3. **Parker ink stain** shows mycelia & spores
(spaghetti & meat balls appearance).

Treatment

I. Topical Therapy

- a. Sodium hyposulphite 30%.
- b. Selenium sulphide 2.5%.
- c. Zinc pyrithione.
- d. Tincture iodine 1-2%.
- e. Imidazole derivatives.
- f. Whitfield ointment.

II. Systemic Therapy *For extensive or recurrent cases*

- a. Ketoconazole 200 mg daily by mouth for 10 days.
- b. Itraconazole.
- c. Fluconazole.

*****Griseofulvin is not effective for yeasts.***

III. To Prevent Relapse

- a. Selenium sulphide shampooing once weekly.
- b. Ketoconazole tablets 3 days per month for 6 months.

b) Candidiasis (Moniliasis)

Candida albicans; a dimorphic organism;
Yeast (Y) form → Commensal; in gut, mouth & vagina.
Mycelial (M) form → Pathogenic.

Predisposing Factors

1. **Trauma**, e.g. friction (obesity).
2. **Moisture & sweating**.
3. **Drugs** as corticosteroids, cytotoxics & antibiotics.
4. **Debilitating diseases** as malignancy or AIDS.
5. **Conditions associated with low resistance**, e.g. DM, pregnancy, Cushing's syndrome & anemia.

Clinical Types

I) Cutaneous Candidiasis

Vesicles that coalesce and rupture → Well-defined, red eroded areas with white fringed edge & forerunners (satellites).

a. Intertrigo

- Groins, axillae & beneath breasts.
- Erosio-Interdigitalis Blastomycetica.
- Angular Cheilitis (Perleche).
- Napkin Dermatitis.

Candidiasis beneath Breast

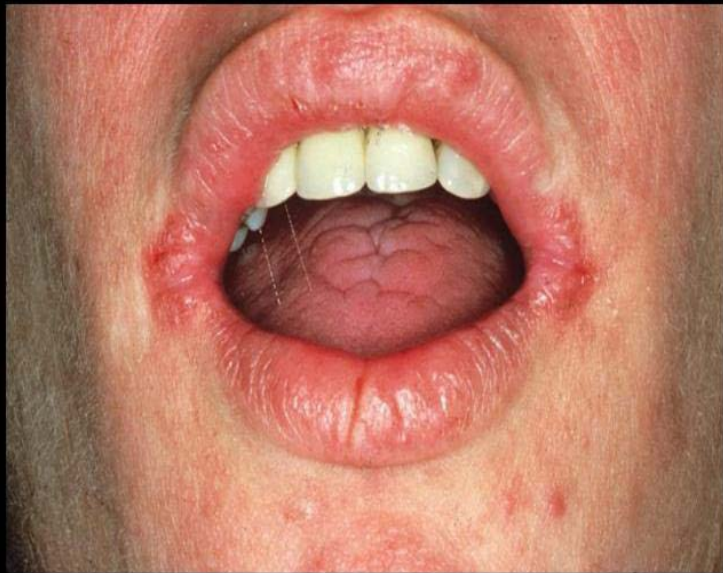


Erosio-Interdigitalis Blastomycetica





Perleche



Bilateral perleche, with cheilitis



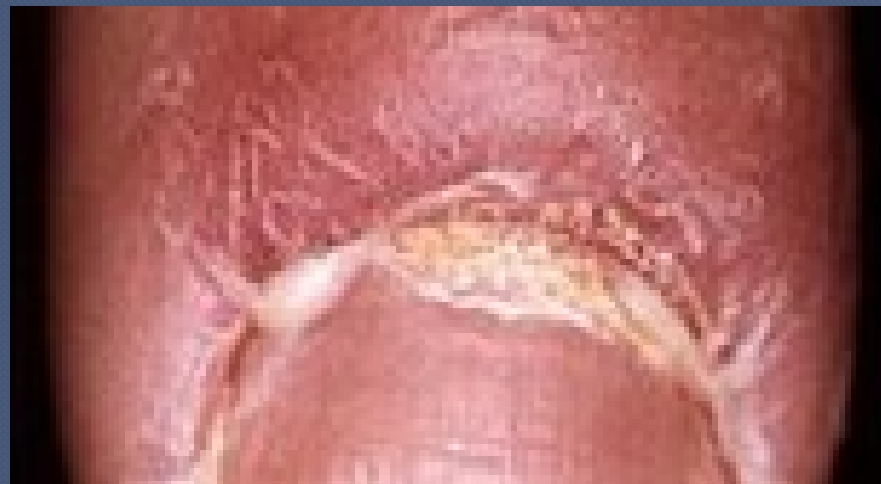
Napkin Candidiasis



b. Paronychia & Onychia

- **Occupation** is a very important predisposing factor.
- **Nail fold** → Swollen, red & slightly tender.
- **Nail plate** → Discoloration, transverse ridging & corrugations.
- **Differential diagnosis** → Pyogenic paronychia.

Paronychia & Onychia



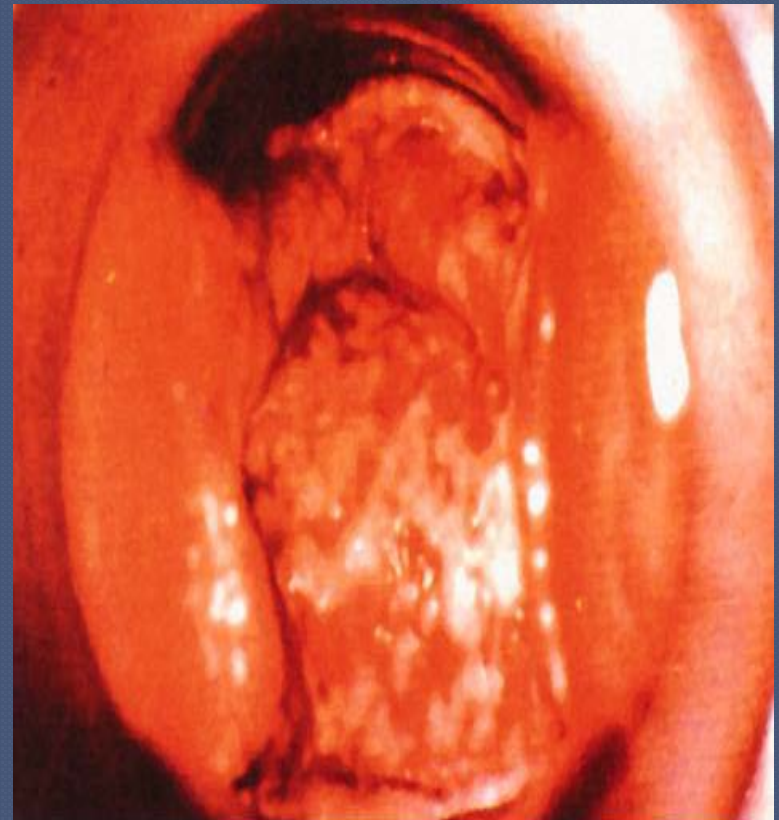
II) Mucosal Candidiasis

- a. Whitish Pseudomembrane (Oral Thrush); in the mouth, in infants & debilitated adults.
- b. Vulvo-Vaginitis (Thrush); pruritus vulvae with a thick, creamy white vaginal discharge; commoner in pregnancy.
- c. Balanitis especially in uncircumcised males.

Oral Thrush



Vulvo-Vaginitis (Thrush)



Balanitis



Treatment of Candidiasis

I) Avoiding Predisposing Factors.

II) Topical Therapy

- a. **Dyes**; castellani's paint & gentian violet 1-2 %.
- b. **Nystatin** creams, powders & vaginal tablets.
- c. **Imidazole derivatives.**

III) Systemic Therapy

- a. **Mycostatin** oral suspension.
- b. **Azoles** as **Ketoconazole** & **Triazoles.**
- c. **Amphotericin (B)** IV infusion in severe cases.

****Griseofulvin is not effective for yeasts.**

ANTIFUNGAL AGENTS

I) Topical Antifungals

a. Paints:

- **Tincture iodine 1-2%** against dermatophytes.
- **Gentian violet**; anticandidal.
- **Castellani paint** against both.

b. Solutions: **Sodium hyposulphite 30%** for TV.

C. Ointments: **Whitfield's ointment**

(salicylic acid 3, benzoic acid 6, lanoline 12 & vaseline add to 100).

d. Creams: *Broadspectrum antifungals.*

- **Azoles** as **clotrimazole** & **miconazole**.
- **Allylamines** as **terbinafine**.

II) Systemic Antifungals

a. Griseofulvin

- Derived from penicillium.
- Fungistatic, effective against dermatophytes.
- Forms: Tablets (125mg) or syrup (125 mg/5ml).
- Dose: 12.5 mg/kg body weight (*maximum dose is 6 tablets*).
- Duration
For tinea capitis → 6-8 weeks.
For tinea circinata → 2-4 weeks.
- Contraindications: Pregnancy & liver diseases.
- Side effects: Hepatotoxicity, photosensitivity & BM depression.

b. Azoles:

- **Broad spectrum antifungals.**
- **Examples:**
 - a. **Ketoconazole:** Hepatotoxicity is a major side effect.
 - b. **Itraconazole:** Minimal side effects.
 - c. **Fluconazole:** Minimal side effects.

c. Allylamines:

- **Effective against dermatophytes only.**
- **Example**
 - Terbinafine:** Minimal side effects.

THANK YOU

IV) PARASITIC INFECTIONS

Intended Learning Outcomes

1. Etiology & C/P of scabies.
2. Differences between animal & human scabies.
3. Treatment of scabies.
4. Different types of pediculosis & their treatment.

a) Scabies

b) Pediculosis

a. Scabies

Types of Scabies

- Human Scabies.
- Animal Scabies.

Human Scabies

Contagious, parasitic disease of the skin

“Sarcoptes scabiei”

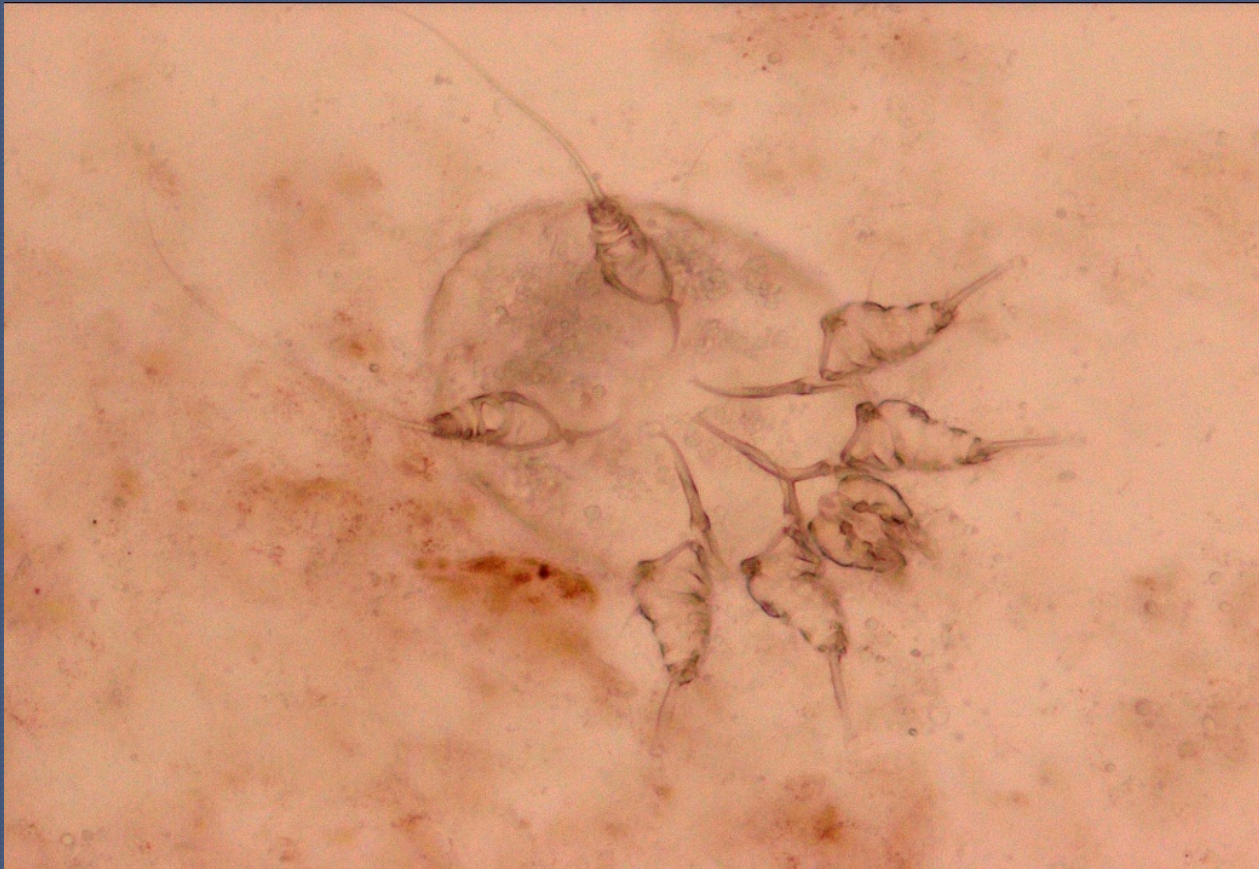
Incubation period

2 weeks.

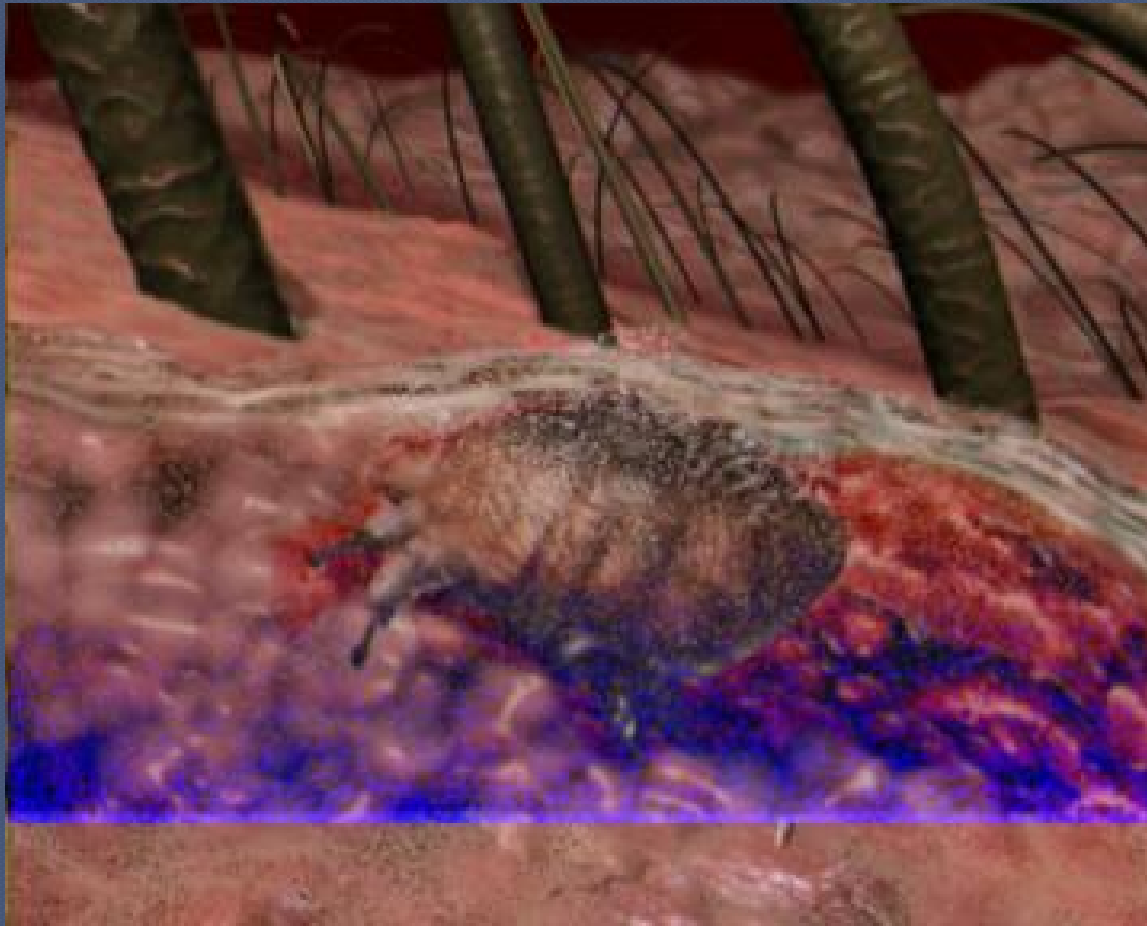
Life Cycle

Fertilized female mite invades the epidermis → Digs a sloping **burrow** → Deposits 2-3 eggs/day to a total of 10-eggs → Female mite dies → Eggs hatch → Nymphs → Surface of the skin → Maturation → Copulation between males & females → New cycle

Sarcoptes Scabiei



Mite Burrowing under the Stratum Corneum



Mode of Infestation

- **Direct** → Close contact with human cases
- **Indirect spread** → Clothes or bedding (*less important*)

*****Mite cannot survive more than a few days away from skin.***

Predisposing Factors

Poor hygiene, overcrowdness & sexual promiscuity.

C/P

- Itching

Most common manifestation & increases at night.

- Burrow

- Linear elevations of the skin, 5-15 mm long.

- Sites

- In-between fingers, wrist area & medial sides of forearms.
- Anterior axillary folds, lower abdomen, breast in females & genitalia in males.
- Medial aspect of thighs & buttocks.
- **Spare** head, neck, upper back, palms & soles
(*diagnostic sign*).

- Other lesions

Furrows, papules, pustules & scratch marks.





Family Members



Post-scabietic nodules

Scabietic nodules

Inflammatory nodules

- Rare condition.
- Inflammatory nodules.
- Hypersensitivity reaction to the parasite.
- Itchy, indurated, reddish-brown in color up to 12 mm.
- **Persist for** weeks or months after ttt of scabies.
- Treated by intralesional steroids.

Nodular Scabies



Scabies in Children & Infants

- Atypical distribution.
- Secondary bacterial infections & eczematous changes.



Complications of Scabies

1. Secondary infections.
2. Insomnia & exhaustion.
3. Acarophobia.

Diagnosis of Scabies

I) Clinical

1. Nocturnal Pruritus.
2. Positive family history.
3. Morphology & distribution of lesions.
4. Spared sites.

II) Investigations

1. Mite is extracted & examined under the microscope.
2. Skin biopsy.

Treatment of Scabies

I) General Instructions

- Treatment of all family members at the same time.
- Disinfection of clothes & beddings.

II) Topical Treatment

After a hot bath, scabicide is applied carefully to all the skin below the neck.

a. Sulphur ppt. Oint. *4 nights*

- 5% for children < 10 years old & 10% for adults.
- Safe in infants.
- Irritant, messy, staining & odoriferous.

b. Crotamiton Cream or Lot. *3 nights*

- 10% preparation.
- Safe in infants.
- Weak antiscabietic; acts more as an antipruritic.

II) Topical Treatment (cont.)

c. Permethrin Cream 1 night

- 5% preparation.
- Safe in infants & pregnant women.
- Has a high cure rate.

d. Benzyl Benzoate Emulsion 3 nights

- 25% preparation.
- Irritant.

e. Gamma Benzene Hexachloride Lot. 1 night

- 1% preparation.
- Irritant & toxic.

III) Systemic Treatment

- a. **Antihistaminics** alleviate itching.
- b. **Anthelmintics**, e.g. **Ivermectin**.
- c. **Antibiotics** treat secondary infections.

Animal Scabies

- **Caused by a different species.**
- **Transmitted from animals to humans, but not from human to human.**
- **Short IP.**
- **Involves sites of contact with the animal.**
- **No burrows; urticarial lesions may be present.**
- **Self-limiting & of a short duration.**

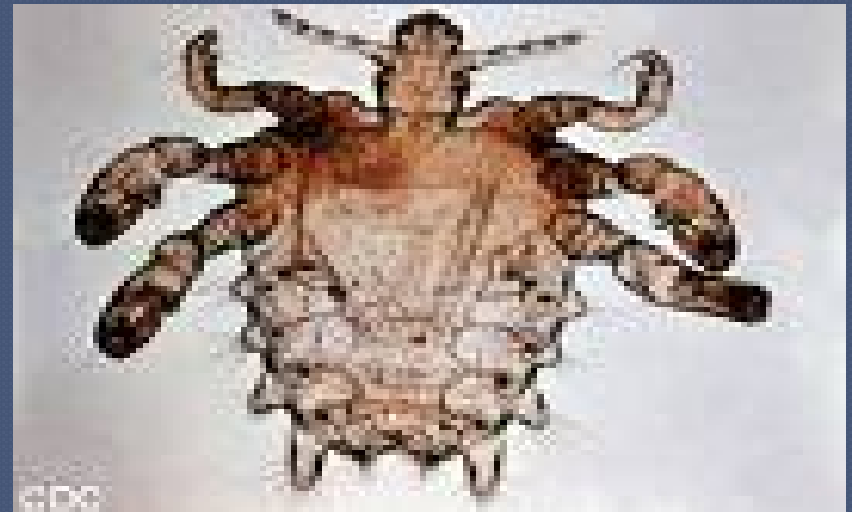
b) PEDICULOSIS

Caused by sucking lice.

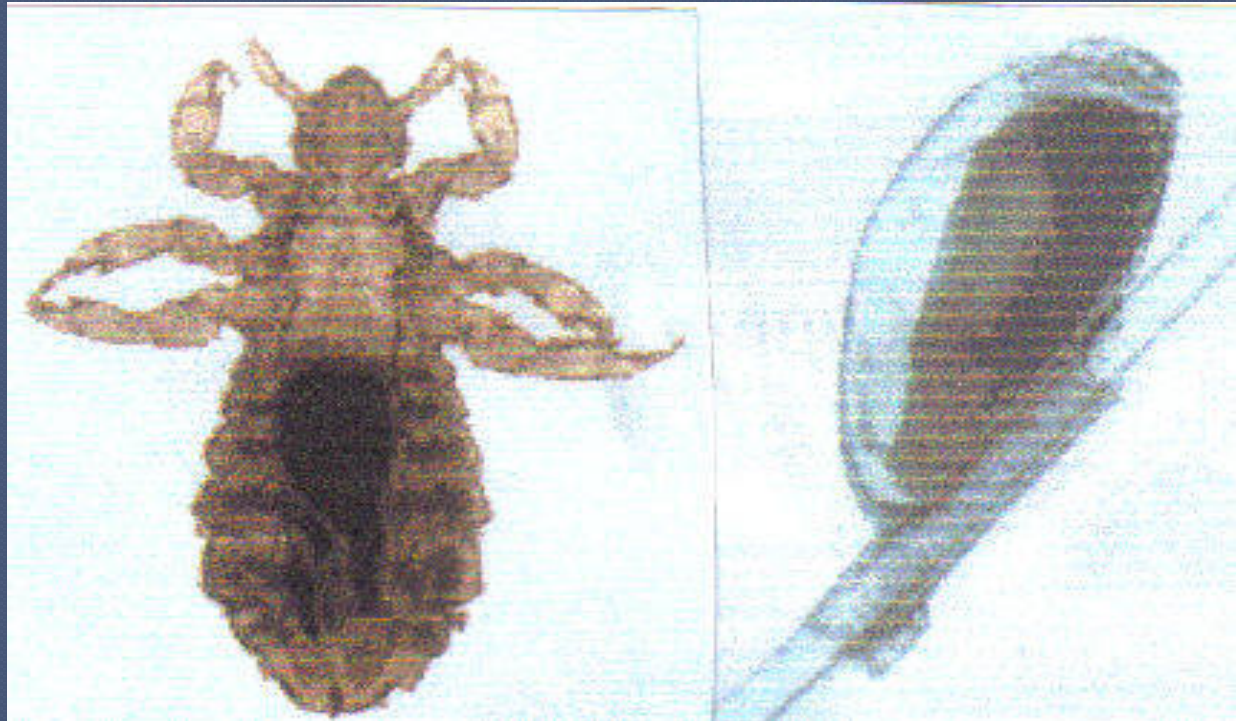
Types

- **Phthiriasis Pubis → Phthirus pubis.**
- **Pediculosis Capitis → Pediculus humanus capitis.**
- **Pediculosis Corporis → Pediculus humanus corporis.**

Phthirus Pubis



Pediculus Humanus Capitis



Pediculus Humanus Corporis



	Phthiriasis Pubis	Pediculosis Capitis	Pediculosis Corporis
Epidemiology	Promiscuity & standards of hygiene.	Preschool & school years.	Personal hygiene.
Mode of Infestation	. Sexual contact. . Clothes or towels.	. Shared hats, caps, brushes or combs. . Close contact.	. Clothes or bedding.
Sites	. Pubic area, lower abdomen & upper thighs.	. Scalp.	. Trunk.
Symptom	. Pruritus.	. Pruritus.	. Pruritus.
Signs	. Nits stuck to hair.	. Nits firmly attached to hair by a cement substance.	. Eggs on seams of clothes or attached to body hairs.

	Phthiriasis Pubis	Pediculosis Capitis	Pediculosis Corporis
Complications	<ul style="list-style-type: none"> •Secondary infection. •Eczematization. 	<ul style="list-style-type: none"> • Impetigo & occipital adenitis. 	<ul style="list-style-type: none"> •Secondary infection.
Treatment	Same as scabies.	<ul style="list-style-type: none"> •Systemic antibiotics. •Antiscabietics <i>Permethrin 2.5%.</i> <i>Gamma benzene hexachloride 1%.</i> •3% acetic acid followed by combing. 	Same as scabies.

Pediculosis Capitis (Nits)



Pediculosis Corporis



THANK YOU

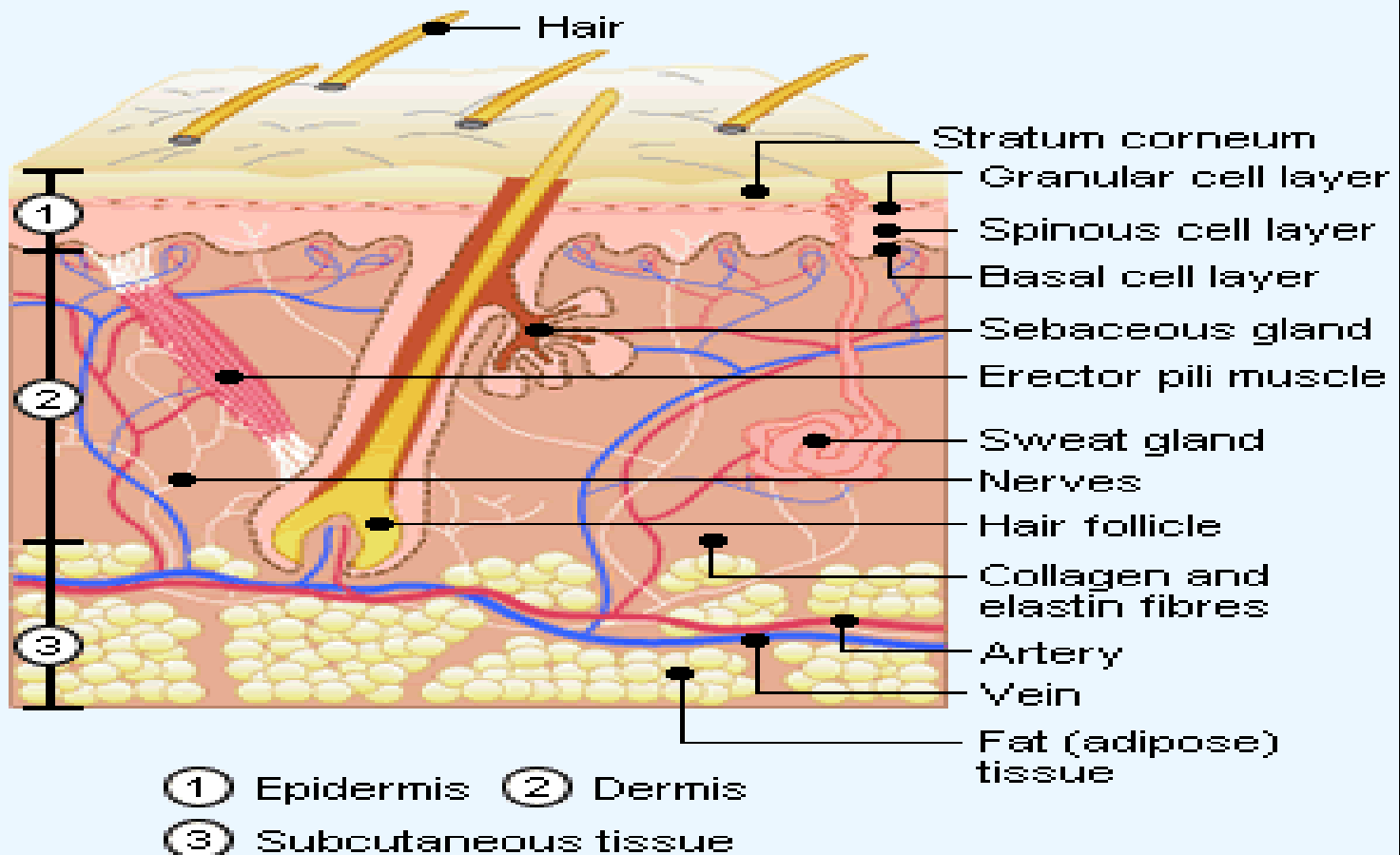
DISORDERS OF SEBACEOUS GLANDS

ACNE

Intended Learning Outcomes

- 1. Nature & pathogenesis of acne.**
- 2. C/P of acne.**
- 3. Treatment modalities of mild, moderate & severe acne.**

HUMAN SKIN



- Chronic inflammatory disorder of **pilosebaceous units**.
- One of the most frequent chronic skin diseases & the commonest dermatological disorder in **adolescents**.
- Primary lesion is the **comedone** (*black head in the pilosebaceous orifice*).
- Other lesions include **papules, pustules, nodules & cysts**.

- Acne is a multifactorial disease.

- Pathophysiologic factors include

- Hyperproliferation of KCs.
- Increase in sebum secretion.
- Change in sebum composition.
- Microbial colonization of pilosebaceous units specially propionobacterium acnes.

Pathogenesis

- Retained **KCs** block follicular opening → Dilatation of lower part of follicle by entrapped **sebum** → Disruption of follicular epithelium → Discharge of follicular content
 - Combination of **keratin, sebum & microorganisms** especially **propionobacterium acnes** → Inflammation & formation of acne lesions

C/P

- Occurs at puberty.
- Involves face, back, chest & shoulders.
- Lesions are polymorphic; primary lesion is the **comedone**.
- Other lesions include **papules, pustules, nodules & cysts**.

Comedones



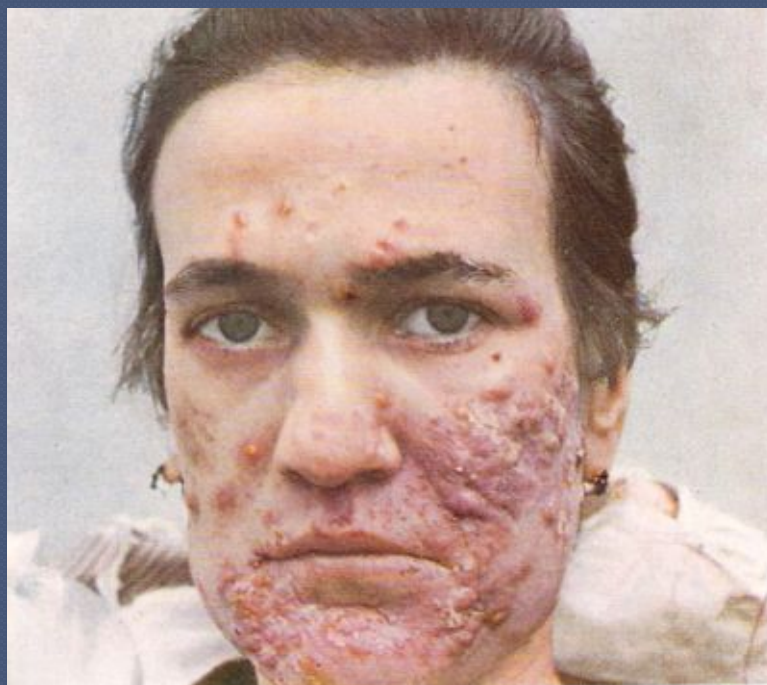
Classification

According to severity, acne is classified into

1. **Mild acne → Comedones + few or no papules.**
2. **Moderate acne → Comedones + papules & pustules.**
3. **Severe acne → Nodules + cysts**
(Acne Conglobata).







Treatment

- Topical application for mild acne.
- Topical & systemic therapy for moderate acne.
- Systemic drugs for severe acne.

I. Topical Therapy

- a. Antibiotics, e.g. erythromycin & clindamycin.
- b. Retinoids decrease keratinization of the orifice.
- c. Benzoyl peroxide 2.5-10% has an antibacterial effect.

II. Systemic Therapy

- a. **Antibiotics**, e.g. **tetracycline**; also has anti-inflammatory actions.

- b. **Retinoids** regulate proliferation of epidermal components (***nodulocystic acne***).

THANK YOU

SCALY ERYTHEMATOUS ERUPTIONS

I) PSORIASIS

Intended Learning Outcomes

- 1. Definition, C/P & clinical types of psoriasis.**
- 2. Treatment of psoriasis.**

- A common genetically determined, hyperproliferative, scaly, erythematous skin disease.
- Less severe in summer than in winter.

Pathogenesis

Increased rate of division of basal cell layer

→ Increased rate of epidermal turnover

with decrease of epidermal turnover time from

28 days → 7 days

C/P

- Primary lesion is a well-defined erythematous **papule** covered by **shiny silvery dry loosely attached scales**. Papules coalesce to give **plaques**.
- Removal of the scales → Appearance of bleeding spots corresponding to tips of dermal papillae
(Auspitz sign; pathognomonic of psoriasis).

Auspitz sign



Clinical Types

I) Psoriasis Vulgaris *most common type*

- Bilateral, symmetrical & very well defined scaly erythematous plaques.
- Involves extensors of upper & lower limbs, elbows, knees, palms, soles, lumbosacral, scalp, flexures, nails & glans.
- Positive **Koebner's phenomenon** (*appearance of primary lesions of the disease at the sites of mechanical trauma*).

Psoriasis Vulgaris













Koebner's Phenomenon



II) Erythrodermic Psoriasis

- Involves more than 90% of the body surface area.

III) Pustular Psoriasis

- Sterile pustules are formed.

IV) Arthropathic Psoriasis

- Psoriasis + Arthritis.

Treatment of Psoriasis

- Reassurance & emotional support.
- Treatment depends upon age, sex, occupation, type & extent of psoriasis.

I) Local Therapy

For mild & localized cases.

a. Corticosteroids

- For localized areas, ointment or lotion (*for scalp*).
- Action increases under occlusion.

b. Salicylic Acid 5% ointment

- Keratolytic.

c. Tar Preparations

- 2-5%, followed by sun exposure.
- Should not be used on face, genitalia or flexures.
- Should not be used in pustular psoriasis.

d. Calcipotriol

Vitamin D3 analogue

- Induces differentiation of KCs.
- Inhibits T-cell proliferation.

e. Phototherapy

- PUVA (*Topical Psoralen + Ultraviolet A*).
- Narrow Band-UVB.

II) Systemic Therapy

For extensive psoriasis vulgaris, erythrodermic, pustular or arthropathic psoriasis.

a. Methotrexate.

b. Retinoids (Acitretin).

c. Cyclosporine.

d. PUVA (oral Psoralen followed in 2 hrs by UVA).

THANK YOU

II) LICHEN PLANUS (LP)

Intended Learning Outcomes

- 1. Definition, C/P & clinical types of LP.**
- 2. Treatment of localized LP.**

Pruritic non-infective scaly erythematous disease of skin, hair & mucous membranes of unknown etiology.

C/P

- Well-defined, flat-topped, polyangular, violaceous & itchy papule with a shiny surface & adherent scales.
- Flexor surfaces especially wrists, flanks, medial thighs, shins of tibia, glans penis, nails, scalp & oral mucosa.
- Pruritus; **rubbing** than scratching.
- **Koebner's phenomenon (*isomorphic response*)**.
- After lesions subside, post lichen hyperpigmentation occurs.

Lichen Planus









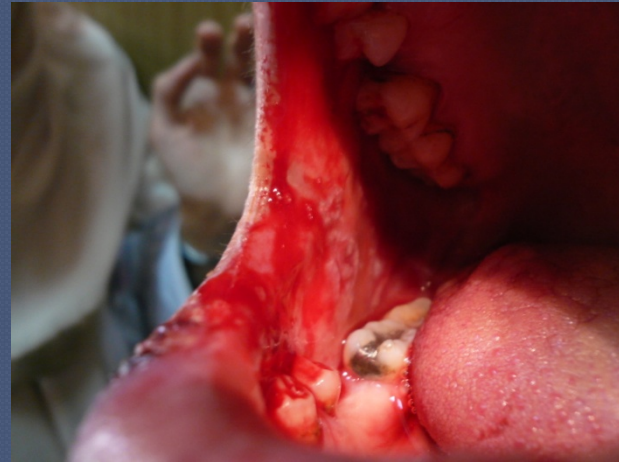
Koebner's Phenomenon



LP of Scalp → Cicatricial Alopecia



LP of oral mucosa



LP of the Tongue



Complications of LP

1. **Squamous cell carcinoma** in oral ulcerative lesions.
2. **Cicatricial alopecia** in scalp LP.
3. **Postinflammatory hyperpigmentation.**

Treatment of LP

I) Local Therapy: Steroids.

II) Systemic Therapy: Steroids & antihistamines (*for itching*).

THANK YOU

III) LUPUS ERYTHEMATOSUS (LE)

Intended Learning Outcomes

- 1. Skin manifestations of systemic LE.**
- 2. Definition & description of discoid LE.**
- 3. Treatment of discoid LE.**

- **An autoimmune collagen disease.**
-

- **Three forms**

1. **Discoid lupus erythematosus (DLE).**
2. **Subacute lupus erythematosus (SCLE).**
3. **Systemic lupus erythematosus (SLE).**

Discoid Lupus Erythematosus

- Chronic inflammatory scaly erythematous eruption confined to the skin.
- Occurs in the 3rd & 4th decades with a female: male ratio of 2:1.
- C/P *Sun-exposed areas*

Well-defined **erythematous** plaques covered with **adherent scales**; lying underneath are **dilated pilosebaceous orifices** (*stippling sign*). Borders of the plaque show **telangiectasia** → After several months, lesions flatten leaving a **thin atrophic scar**

DLE











Treatment of DLE

1. Avoid sun exposure.
2. Sun-screens.
3. Systemic photoprotectives.
4. Topical corticosteroids.
5. Intralesional corticosteroids.
6. Systemic corticosteroids.

Systemic Lupus Erythematosus

- A systemic disease affecting multiple organs.
- Immunological abnormalities & pathological changes.
- Occurs in early adult life with a female: male ratio of 8:1.

C/P

Skin Manifestations

- Discoid-like lesions.
- Erythematous, non-scarring patches on light-exposed areas.
- Purpura & vasculitis.
- Alopecia; diffuse & non-cicatricial.
- Raynaud's phenomenon.





THANK YOU

ALLERGIC DERMATOSES

I) ECZEMA (DERMATITIS)

Intended Learning Outcomes

- 1. Clinical classification & most common types of eczema.**
- 2. C/P of contact dermatitis & atopic dermatitis.**
- 3. Diagnostic tools & general treatment of eczema.**

Ill-defined erythema, itching & vesicle formation.

Clinical Types

a) Acute Eczema

Erythema, swelling, vesicles & oozing → Crusting.

b) Chronic Eczema

Lichenification, excoriations & hyper-or hypopigmentation.

c) Subacute Eczema

Features of both.

Main Types of Eczema

I) Contact Dermatitis

- a. Primary Irritant Dermatitis.**
- b. Allergic Contact Dermatitis.**

II) Atopic Eczema.

I) Contact Dermatitis

a. Primary Irritant Dermatitis

- Any individual; previous contact is not required.
- Soon after exposure.
- Direct damage by strong acids or alkalis or cumulative damage by mild irritants.
- Soaps, detergents, vegetables or solvents.
- Compulsive washers, housewives, dishwashers, nurses & surgeons.

Irritant CD





b. Allergic Contact Dermatitis

- Immunological reaction that develops in genetically susceptible individuals after exposure to allergen.
 - Presentation of allergen by LCs to T-cells → Re-exposure to same antigen → Lesions develop in sensitized individuals at sites of contact
- Nickel, chromate, rubber, resins, glues, cleansers, cosmetics & medications (*sulfa powder, penicillin ointment & local antihistamines*).

Allergic CD









Allergic CD to Airborne Allergen



Diagnosis of Contact Dermatitis

1. History.
2. Clinical picture.
3. Histopathology.
4. Patch testing.



II) Atopic Eczema

Atopy

Genetic hereditary predisposition to develop hay fever, bronchial asthma, allergic rhinitis or atopic dermatitis.

Pathogenesis

Triggering factors → T-helper cell proliferation →

Cytokine production → Pathological & clinical changes

Ppt Factors

Irritants, allergens (*house-dust mite*), excessive washing, food, staphylococci, viruses, cold weather & lack of humidity.

Phases

a. Infantile Phase

Infantile Eczema

- 2 months-2 years.
- Acute eczema of cheeks & dorsa of hands or may involve whole body.

b. Childhood Phase

- 4-12 years.
- Groups of itchy papules involve the flexures particularly the antecubital & popliteal fossae & sides of the neck.

c. Adult Phase

- Over 12 years.
- Similar to childhood type + hyperpigmentation & lichenification.

Infantile AD





Childhood AD



Adult AD



AD in flexures



Treatment of Eczema *Avoid the causative agent*

a) Acute Eczema

I) Local

- Drying antiseptic lotions (*aluminum acetate, KMnO_4 1/8000 or normal saline*).
- Corticosteroid creams.

II) Systemic

- Antihistamines.
- Corticosteroids.

b) Chronic Eczema

I) Local

- Corticosteroid ointments.

II) Systemic

- Corticosteroids.

****In Atopic Dermatitis**

- 1. Patient & family education.**
- 2. Avoidance of stress, cold, dryness & irritants.**
- 3. Frequent application of moisturizers & emollients.**
- 4. Topical immunomodulators.**
- 5. NB-UVB in severe cases.**

THANK YOU

II) URTICARIA

Intended Learning Outcomes

- 1. Definition & description of urticaria.**
- 2. Mechanism of urticaria.**
- 3. Classification & different types of urticaria.**
- 4. Treatment of urticaria & angioedema.**

-
- A common allergic skin disease.
 - Type I hypersensitivity reaction.
 - Release of **histamine** leads to vasodilatation with local increase of permeability & development of a transient edema of the skin; **wheal**.
 - Itchy sensation.

C/P of Wheals

- Sudden appearance of whitish or reddish slightly elevated edematous lesions.
- Size varies from few mms-several cms.
- Any area of skin surface or mucous membranes.
- Localized or more commonly generalized.
- **Evanescent.**
- If lesions continue to erupt for more than 3 months, it is called **chronic urticaria.**

Pathogenesis of Urticaria

- *On first antigen exposure*, IgE antibodies are formed



Bind to IgE receptors on mast cells

- *On second exposure* (after one week or more) *to the same antigen or antigenically-related compound*,

ag-ab reaction occurs on surface of mast cells



Degranulation of mast cells & release of chemical mediators (*histamine, prostaglandins, heparin, ...*)

Urticarial Wheal



Urticarial Wheals





Etiology of Urticaria

1) Exogenous Causes

a. Ingestants

- **Foods** as fish, milk, eggs, chocolates, strawberry, banana, nuts & food preservatives.
- **Drugs** as salicylates, penicillin, sulfonamides & NSAIDs.

b. Injectants

- **Blood elements.**
- **Drugs**, e.g. penicillin, other antibiotics & NSAIDs.
- **Insect bites**, e.g. mosquitoes, fleas & ants.

c. Inhalants

- **Grass pollens.**
- **Mould spores.**
- **Perfumes.**

2) Endogenous Causes

- a. Septic foci: Bacterial, viral or fungal.
- b. Intestinal parasites: Protozoal & helminthic infestations.
- c. Medical disorders: SLE, liver diseases, malaria & thyrotoxicosis.
- d. Internal malignancies: Lymphoma or GIT cancer.
- e. Pregnancy.

Clinical Types of Urticaria

I) Ordinary Type.

II) Angioedema

- Vasodilatation of larger-sized blood vessels of SC tissue → Edema.
- Soft tissues as lips, eyelids & genitals.
- Laryngeal affection → Edema of vocal cords & suffocation.

Angioedema





Clinica Dermatologica - UNINA "Federico II"

III) Cholinergic Urticaria

- Patient feels prickly & itchy sensation after sweating.
- Scalp, neck & upper chest more than other body areas.
- Small wheals may be observed corresponding to sweat glands.
- Lesions subside usually within one hour.
- Acetylcholine is the mediator.

IV) Physical Urticaria

- Wheals appear at sites of exposure to **physical agents**.
- Types
 - **Solar Urticaria** → Sun exposed parts.
 - **Pressure Urticaria** → Prolonged pressure.
 - **Cold Urticaria** → Cold.
 - **Heat Urticaria** → Heat.

Cold Urticaria



V) Papular Urticaria

- Occurs in response to **insect bites**.
- Usually affects **infants & children**.
- Involves exposed areas if due to flying insects (mosquito) or covered areas if due to non-flying insects (*ants & fleas*).
- A wheal appears at the site of the bite → Within one or two days, an itchy erythematous **papule** appears at the center → Stays for few days (\pm **vesicle**).
- Insect injects more than one antigen. This leads to **two types** of allergic reactions;
 - **Type I** → Antigen leads to wheal formation.
 - **Type IV** → Another antigen leads to lymphocytic cell infiltration & papule formation.

Papular urticaria





Treatment of Urticaria

I) Avoid the cause if known.

II) Local Therapy

- a. **Cold compresses** cause vasoconstriction.
- b. **Calamine lotion** has a soothing effect.
- c. **Corticosteroids** in localized forms as papular urticaria.

III) Systemic Therapy

- a. **Antihistamines.**
- b. **Corticosteroids.**
- c. **Adrenaline**, in laryngeal angioedema, 0.2-0.5 ml of 1:1000 solution, SC or IM (*never IV*).

THANK YOU

IV) DRUG ERUPTIONS

Intended Learning Outcomes

- **Basic information about most common forms of drug eruptions.**

-
- May manifest in several forms which may mimic any skin disease.
 - **Characterized by**
 - An acute onset.
 - Atypical distribution.
 - More inflammation.
 - Subsidence after stoppage of causative drug.

Common Drug Reactions Include

1. **Urticaria & Angioedema.**
2. **Acneform Eruptions;** most commonly caused by corticosteroids.

3. Fixed Drug Eruption

- Most commonly caused by **sulfonamides & NSAIDs**.
- Called "**fixed**" because it is
 - Fixed** to the drug
 - Fixed** to the site&
- **Characterized by** a permanganate-colored macule or patch → May progress to bullae followed by rupture & formation of an erosion.
- Any part of the skin or mucous membranes, but the most common sites are lips & genitalia.

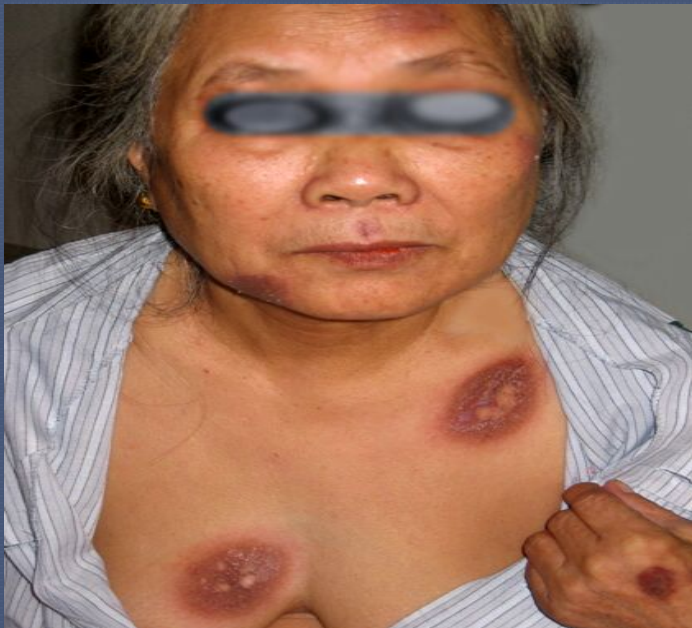
Angioedema



Acneform Eruption



Fixed Drug Eruption













THANK YOU

DISORDERS OF MELANOCYTES

VITILIGO

Intended Learning Outcomes

- 1. Definition & description of vitiligo.**
- 2. Etiology & types of vitiligo.**
- 3. Treatment of vitiligo.**

- A common, non-infectious, genetically determined disorder.
-
- Characterized by **loss of melanocytes**.
 - Primary lesion is a well circumscribed milky white **macule** or **patch**.
 - Affects any part of the skin.
 - Melanocytes are destroyed & disappear from epidermis



No melanin production



Milky white **macules** & **patches**.

Vitiligo in different sites





Clinical Types

1. **Focal Type**; one or few patches involved.
2. **Unilateral Type**; lesions stop at the middle-line.
3. **Generalized Type**; lesions are scattered all over body.
4. **Universal Type**; all skin surfaces are involved.

Focal Vitiligo



Unilateral Vitiligo

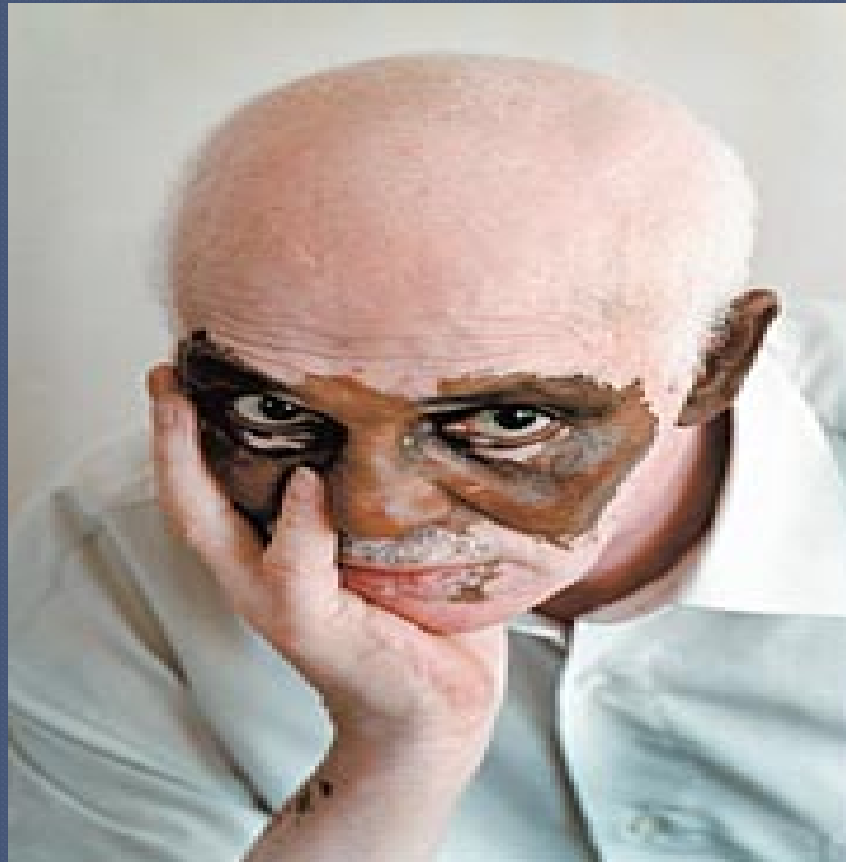


Generalized Vitiligo



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Universal Vitiligo



Etiology

1. Autoimmune Theory

Antimelanocyte antibodies → Destruction of melanocytes.

2. Neurogenic Theory

**Melanocytotoxic substances released from nerve endings
→ Destruction of melanocytes.**

Treatment

I) Phototherapy

Stimulates residual melanocytes in hair follicles.

- a. PUVA: Oral **or** topical **P**soralen + **UVA**.
- b. NB-UVB (311 nm): Lamps without psoralen.

II) Medical Treatment

- a. Steroids; topical & systemic.
- b. Immunomodulators.
- c. Antioxidants.

****In cases of universal vitiligo,**
residual pigmentation of normal color may be
removed by
Phenolic compounds,
Hydroquinone derivatives or
by LASER.

THANK YOU

DISORDERS OF HAIR FOLLICLES

ALOPECIA (HAIR LOSS)

Intended Learning Outcomes

- 1. Definition & types of alopecia.**
- 2. Androgenetic alopecia; definition and summary of etiology & management.**
- 3. Alopecia areata; definition, types, differential diagnosis & treatment.**

Types of Alopecia

a) Cicatricial Alopecia.

b) Non-Cicatricial Alopecia

I) Androgenetic Alopecia (*Familial Baldness*).

II) Alopecia Areata.

a) Cicatricial Alopecia

Hair follicles are destroyed → Scar & permanent hair loss.

Causes

- 1. Mechanical trauma.**
- 2. Burns.**
- 3. Fungal infections, e.g. kerion & favus.**
- 4. Viral infections, e.g. herpes zoster.**
- 5. Collagen diseases, e.g. DLE.**
- 6. Inflammatory diseases, e.g. lichen planus.**
- 7. Neoplasms, e.g. basal cell carcinoma.**



b) Non-Cicatricial Alopecia

l) Androgenetic Alopecia (*Familial Baldness*)

- Pathogenesis

Activity of 5-alpha reductase is accentuated in susceptible follicles → Testosterone is converted to the more potent dihydrotestosterone → Slowing of protein synthesis & **increase hair shedding**

- C/P

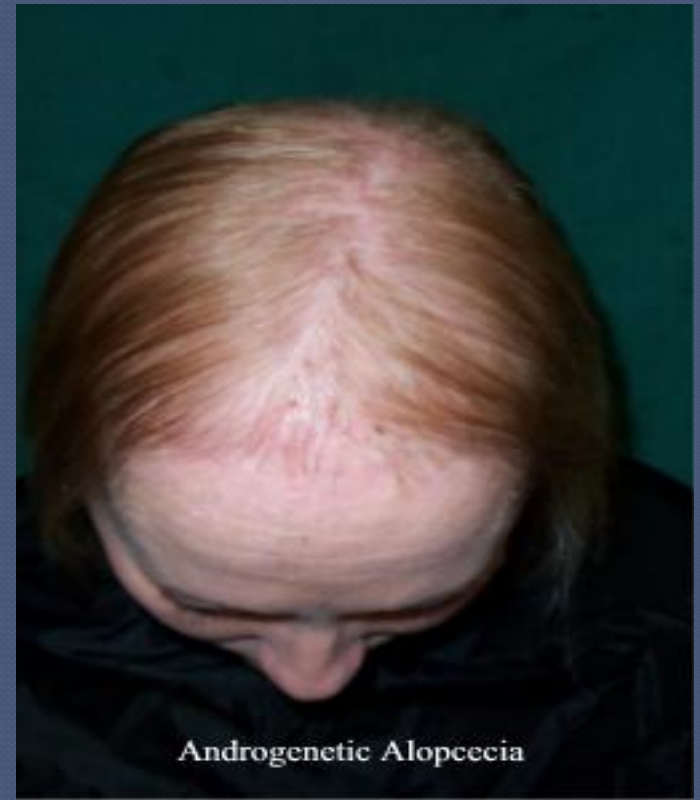
- Males → Begins by frontal & bitemporal recession, and then it involves the vertex.
- Females → Vertex from behind frontal hairline is the target area.

Androgenetic Alopecia

Male



Female



- **Treatment**

1. **2-5% topical minoxidil** increases hair growth.
2. **Inhibitors of 5-alpha-reductase**, e.g. **finasteride** 1 mg/day.
3. **Antiandrogens**, e.g. **cypoterone acetate** & spironolactone; used only in female androgenetic alopecia.

II) Alopecia Areata

- Etiology

- Genetic factors; 10-20% of cases give +ve family history.
- Autoimmune theory.

- Precipitating Factors

Emotional or psychological stresses cause changes in immune function.

- **C/P**

Sudden complete loss of hair with normal skin.

- **Clinical Types**

1. **Patchy type**, the commonest.
2. **Alopecia Totalis**; loss of all scalp hair.
3. **Alopecia Universalis**; loss of all body hairs.

- **Course**

- **Unpredictable; spontaneous remission & exacerbations.**
- **Some cases may persist for long periods.**

Patchy Alopecia Areata



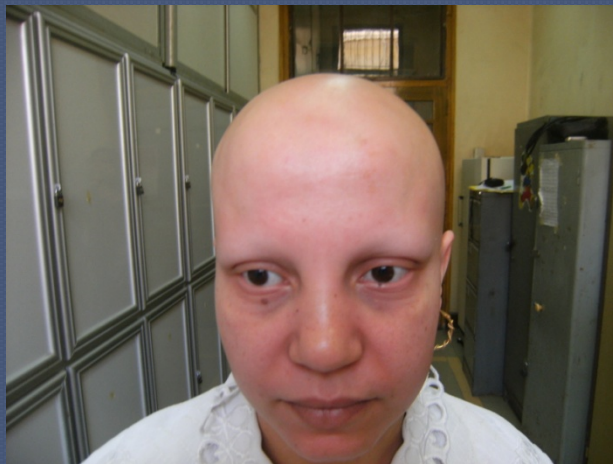






Alopecia Totalis





Alopecia Universalis



- **Treatment**

I) Topical

1. **Local irritants** as **tincture iodine 2-4% & anthralin.**
2. **Corticosteroid** creams, ointments, lotions or intralesional.
3. **PUVA therapy**; local psoralen + UVA light.
4. **2-5% minoxidil.**

II) Systemic

1. **Sedatives & antidepressants.**
2. **Corticosteroids** in alopecia universalis.

THANK YOU